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Archives of Neurology and Psychiatry

Vol. 5

JANUARY, 1921

No. 1

A CLINICAL AND ANATOMIC STUDY OF A VASCULAR LESION OF BOTH CEREBELLAR HEMISPHERES

WITH ESPECIAL REFERENCE TO CEREBELLAR CATALEPSY AND NYSTAGMUS, AND THE ANATOMIC CONNECTIONS OF THE INFERIOR OLIVE *

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CLINICAL STUDY

History.—J. E. S., aged 58, a native of Boston, an inmate of the Relief Home, San Francisco, was first examined by me on Dec. 7, 1911. His illness began with a sudden onset three years before, in Japan, without previous ill health or warning. Seated in a tea house in Yokohama, he experienced a sensation as if he were "struck by a ball of lightning in the knees." He arose and attempted to walk, which he was able to do for a distance of about 25 yards, but would then have fallen had he not been aided. There was no loss of consciousness, no pain, headache, nausea or vomiting. Incontinence of urine immediately followed the attack, and later there was imperative micturition. Following the stroke he remained in bed several days. It was at once apparent that control of his left arm and leg was practically lost for all voluntary movements. The right arm and leg were also affected but to a lesser extent. Being right-handed, he could feed himself but with considerable difficulty. It was not possible for him to walk or even to stand without support, and he would sit all day in a wheel chair. His speech had changed, having become slow and deliberate without any disturbance of pronunciation. Later, hearing and vision were affected; vision was affected for near objects but not for distance. The patient believed that his mentality was as good as formerly, that his memory was as good, and that his disposition had not changed. He was not emotional. There had been no remarkable change in his condition in the three years that had elapsed. He did not complain of weakness, and with the exception of his great loss of muscular control and imperative micturition, he considered himself in average health.

The family and previous personal history of the patient were unimportant. Well as a child, his first serious illness was an attack of typhoid in 1898. Venereal infection was denied. He was a total abstainer from alcohol, but smoked in moderation.

Examination.—Examination revealed a man apparently of the given age, practically helpless because of great motor incoordination in all extremities

* Read at the Forty-Sixth Annual Meeting of the American Neurological Association, New York, June, 1920.

* From the Neurological Clinic, Department of Medicine, Leland Stanford Junior University, San Francisco.

but especially in the left extremities. To walk was impossible and even to attempt it was to risk a fall. The patient could scarcely stand by supporting himself with both hands on the bed rail. He would sit upright in his wheel chair for hours at a time with no discomfort or difficulty, and he spent his days in this manner. Intelligence and emotions were evidently not impaired. Speech was typically scanning, but there was no dysarthria.

An analysis of his disturbed coordination was attempted. In the different tests the incoordination was of the same character on both sides but far greater on the left. When the patient was directed to place his index finger on the tip of his nose he would always pass it, sometimes moving his hand several times back and forth in a jerky awkward fashion, giving the impression that the orientation was unimpaired. This was supported by the fact that the same errors were made with the eyes open or shut. The same phenomena was found in the heel to knee test. This failure of attaining the designated object was interpreted as a *mouvement démesuré* (Thomas and Jumentié) and was probably dependent on disturbed muscle synergy.

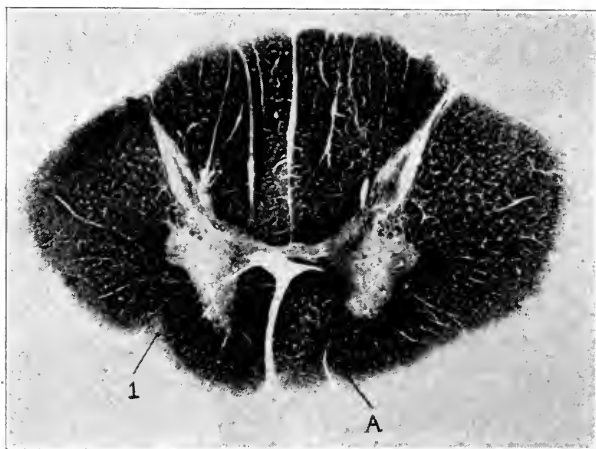


Fig. 1.—Mid-Cervical cord: 1, degeneration of left Helweg's bundle; A, degeneration in the right anterior column. In this and in the following illustrations the left side of the figures corresponds to the left side of the brain.

Disturbance of synergy in this patient was examined by Babinski tests, which would tend to dissociate muscular movements. When the patient in the sitting position was directed to touch with his foot the examiner's hand held at about the level of the trunk in front of him, there would at first be a flexion of the thigh on the pelvis and then an extension of the leg, the foot finally attaining the mark in this fashion. In the upper extremities the alternate supination and pronation of the hand revealed great deficiency in the rapid and accurate successive contractions of antagonistic muscles; there were *adiadokokinesis* in the left hand, causing wide excursion of the forearm, and *dysdiadokokinesis* in the right.

1. Babinski: De l'équilibre volitionnel statique et de l'équilibre volitionnel cinétique, *Rev. Neurol.* 10:470, 1902.

Perhaps the most striking symptom in this case was that of cerebellar catalepsy (Babinski). When the patient lay on his back the thighs flexed on the pelvis and somewhat abducted and the legs on the thighs, after a few unsteady movements of the extremities, a remarkable immobility ensued. Not only was this immobile posture noteworthy in one who showed such great disturbance of voluntary motion, but it was actually more pronounced than in the normal case, and the duration was longer. This symptom was tested repeatedly and found to be constant.

It was not noted that the patient had a tendency to fall in any one direction when he lost his equilibrium, that there was hypotonia present or tendency to fixed attitudes. The muscular force of the extremities was well preserved.

Voltaic vertigo was tested from the standpoint of the functional labyrinth tests. With an interrupted current of from 14 to 16 ma., with both electrodes above and in front of the tragus, a slight inclination of the head was produced toward the side of the positive pole. Turning tests or syringing were

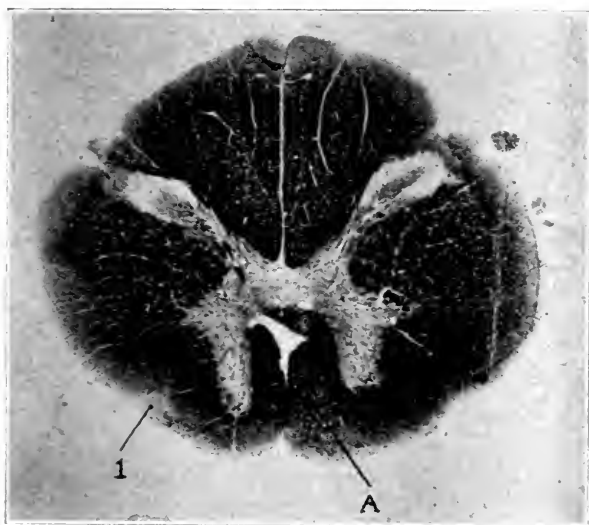


Fig. 2.—Upper cervical cord: 1, degeneration of left Helweg's bundle; A, degeneration in right anterior column.

not performed. Spontaneous past pointing after Bárány was tested, but pointing was always done correctly in the sense that allowance being made for dyssynergia, there was no constant tendency to deviate to one side. It is not noted with which portion of the extremities these tests were made.

In the general neurologic examination there were no muscular atrophy, tremor or fibrillations present. It was noted that the left tendon reflexes were increased over those on the right, with the exception of the Achilles' reflexes, which were equal. The Babinski reflex was absent. Sensibility tests for light touch, pain and temperature were normal throughout. The stereognostic sense was intact. Postural sense and osseous sensibility were not impaired.

2. Babinski: Proceedings XVIIth International Congress of Medicine, London, 1913; Section XI, Neuropathology.

Cranial Nerves: The pupils were about equal in size but somewhat irregular in outline and reacted to light, accommodation and convergence. There was no spontaneous nystagmus in looking forward to the extreme right or left, or upward. The movements of the eyeballs were normal, but it seemed to be an effort for the patient to look upward, and he complained of pain in the muscles at the back of the neck when he did so. The corneal reflex was present. Fields of vision were normal by roughly testing. Sensation over the face was normal. Hearing was diminished in both ears to watch tick. There was no facial paralysis. The walls of the soft palate moved equally and well, and the pharyngeal reflex was active. There were no signs of bulbar paralysis. The spinal accessory nerve was normal. The tongue protruded in the mid-line without tremor.

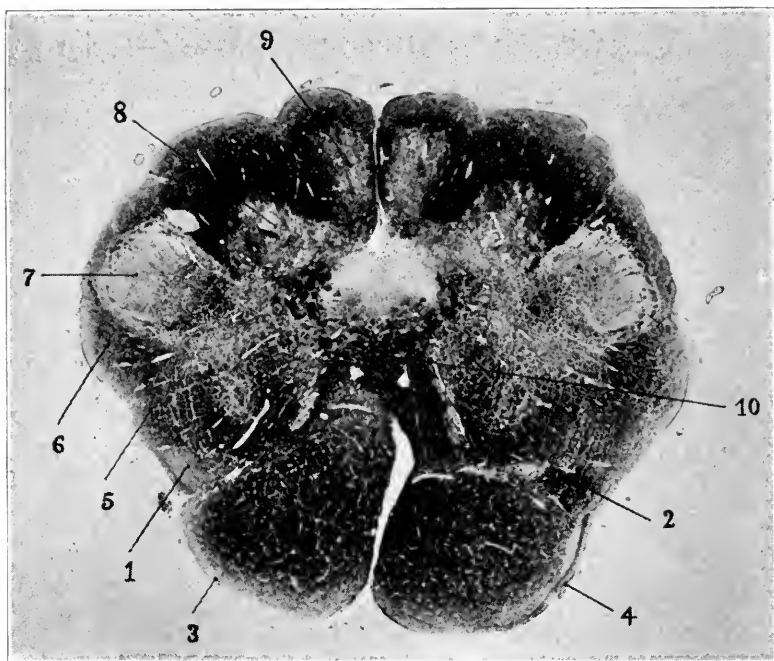


Fig. 3.—Transverse section of medulla at the beginning of the pyramidal decussation: 1, degeneration of left Helweg's bundle; 2, right Helweg's bundle; 3, degeneration of left lateral external arcuate fibers; 4, right lateral external arcuate fibers; 5, Gower's tract; 6, direct cerebellar tract; 7, substantia gelatinosa; 8, nucleus cuneatus; 9, nucleus gracilis; 10, pyramidal decussation.

The patient was well nourished, ate and slept well. The cardiovascular system seemed to function well. The pulse was of fair tension and volume, and the radial artery was not appreciably hardened. The heart was not enlarged, and no bruits were heard. The abdomen and lungs were reported normal, with the exception of a slight emphysema.

Because of the history of sudden onset, the stationary character of the affection and the characteristic cerebellar symptomatology which was predominant on the left side, a diagnosis of a vascular lesion in the left cere-

bellar hemisphere was made. The patient died suddenly on Feb. 15, 1912, and it was suspected that he suffered an apoplectic stroke.

ANATOMIC STUDY

The Lesion.—The brain and cord alone were removed at necropsy so that the exact cause of death was not determined. A marked arteriosclerosis, particularly at the base of the brain, was observed, but no thickening of membranes, adhesions or tumor mass was found. External to the left superior peduncle (Fig. 11, No. 58), a small area of superficial softening was seen; otherwise the external appearance of the brain was normal. The brain was hardened in a 10 per cent. solu-

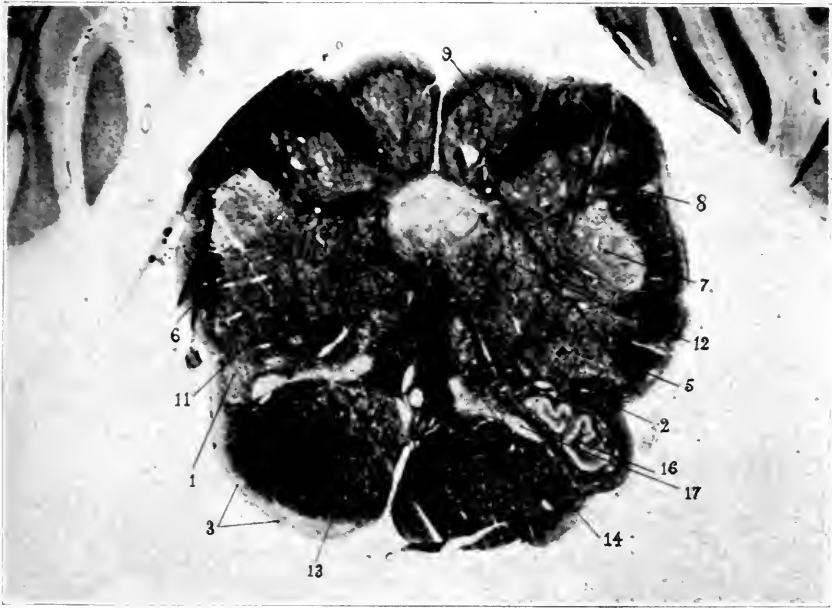


Fig. 4.—Transverse section at caudal extremity of inferior olive: 1, degeneration of left Helweg's bundle; 2, right Helweg's bundle; 3, degeneration of left lateral external arcuate fibers; 5, Gower's tract; 6, direct cerebellar tract; 7, substantia gelatinosa; 8, nucleus cuneatus; 9, nucleus gracilis; 11, left internal arcuate fibers; 12, degeneration of right internal arcuate fibers; 13, degeneration of left median fillet; 14, right median fillet; 16, right inferior olive; 17, medial accessory olive.

tion of formaldehyd before it was cut. It was then studied macroscopically by serial sections; the cerebellum and medulla were removed by a transverse cut through the peduncles, and the hemispheres were divided by cutting through the corpus callosum. Both cerebral hemispheres were then cut horizontally into a number of slices, after the procedure of Marie. No lesions were found. Transverse sections through

the cerebellum, however, revealed old symmetrical areas of softening in the central white of both hemispheres with cavity formation, the destruction of tissue being greater on the left side. In neither hemisphere did the softening reach the periphery. In both hemispheres the lesion extended well backward toward the posterior poles; on the left side (Figs. 8, 9 and 10, Nos. 27 and 28) it destroyed the dentate nucleus, nucleus emboliformis and globosus, and extended well forward toward the anterior pole, as seen by fiber degeneration dorsal and lateral to the superior peduncle (Fig. 11, No. 58). In the right hemisphere the dentate nucleus and accessory nuclei were conserved, but laterally and

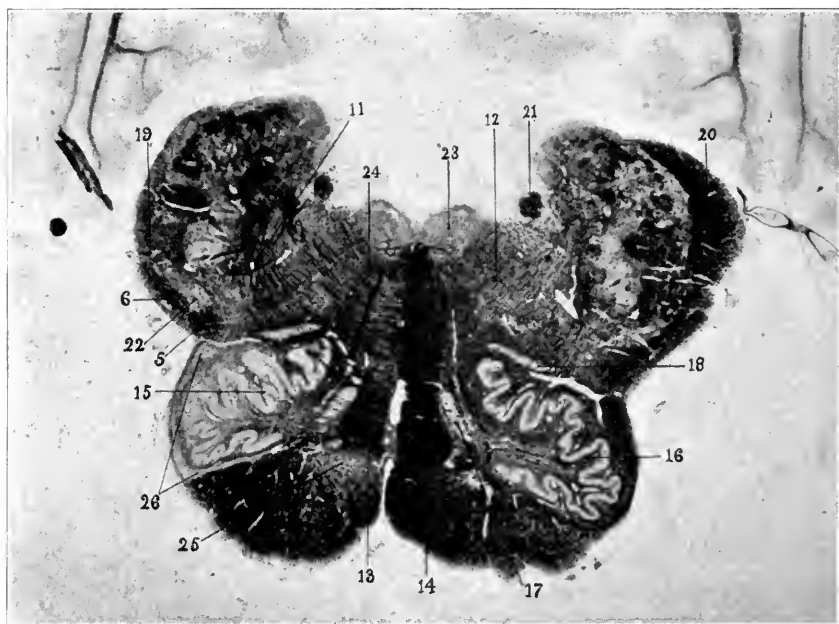


Fig. 5.—Transverse section through lower olive: 5, Gower's tract; 6, direct cerebellar tract; 11, left internal arcuate fibers; 12, degeneration of right internal arcuate fibers; 13, degeneration of left median fillet; 14, right median fillet; 15, left inferior olive; 16, right inferior olive; 17, medial accessory olive; 18, dorsal accessory olive; 19, left restiform body; 20, right restiform body; 21, fasciculus solitarius; 22, lateral nucleus; 23, hypoglossal nucleus; 24, root fibers of hypoglossal nerve; 25, degeneration in left pyramid; 26, peri-olivary degeneration.

below there were seen degenerations which involved the middle cerebellar peduncle. The vermis and roof nuclei were not involved in the destructive process, and no degenerations were present.

In addition to these large lesions, there were three other minor lesions, one mentioned above found external to the left superior peduncle, and another (Fig. 9, No. 49) central and found at its greatest

extent below the sixth nerve nucleus on the left side. The latter reached to the midline, not measuring over 1.5 mm. at its greatest transverse diameter. It did not extend below the upper level of the inferior olive, and traced upward it was seen to disappear at the crossing of the trapezoid body, a number of whose fibers it divided. The third small lesion (Fig. 12, No. 68), a degeneration in the right crusta, was lost in lower sections and traced upward finally disappeared at the level of the upper end of the red nucleus. This degeneration was due to a local softening from thrombosis of smaller vessels.

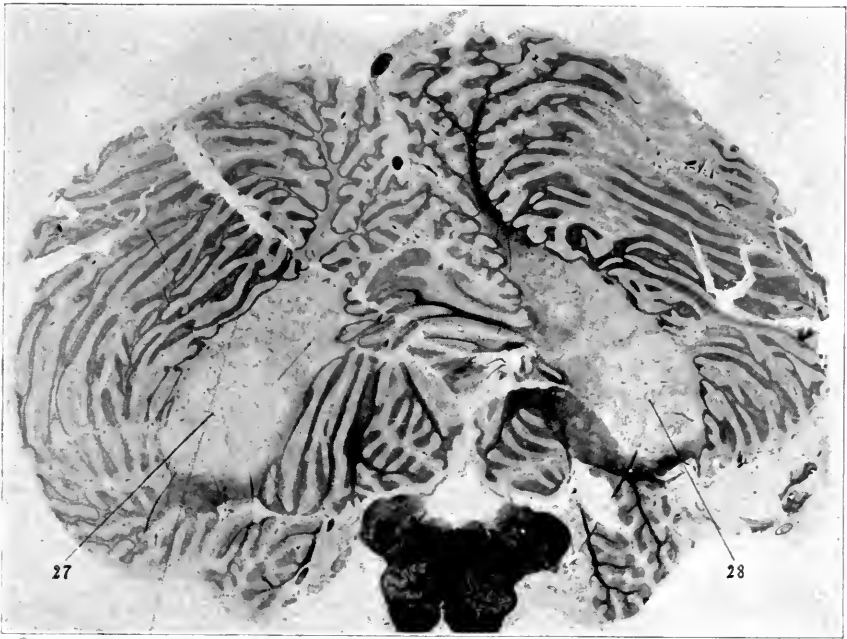


Fig. 6.—Transverse section of posterior cerebellum and medulla: 27, softening in left cerebellar hemisphere; 28, softening in right cerebellar hemisphere.

Fixation and Staining.—The entire brain and cord were hardened in bichromate solution. The rhombencephalon and portions of the upper cervical and dorsal and lumbar cords were included in celloidin, blocked and cut in serial transverse sections and stained by the Kul-schitzky and Weigert methods, alternate sections being counterstained by cochineal. Subsequently, the peduncular and subthalamic regions were reconstructed in one block, and the basal ganglions in another, so that eventually three separate blocks were serially sectioned, including all parts of the brain except the upper cortex, frontal and occipital poles. Due to the extended study of these different blocks and the interruption occasioned by the World War, the final report of this

case was delayed until the present time. Mention is made of this case, however, in the *California State Journal* for July, 1913, and in the *Journal of Nervous and Mental Diseases* for May, 1915.

The Degenerations.—Striking degenerations occasioned by the cerebellar lesions were the practically complete degeneration of the left superior peduncle, degeneration of the right red nucleus and in Forel's field, degeneration of the left restiform body and atrophy of the right inferior olive. The atrophy of the right olive was evidenced by its small size, principally due to diminution in width of the cellular layer and its poverty of cells. However, both the internal fibers entering the



Fig. 7.—Enlargement of medulla in Figure 6. Section at emergence of vagus nerve: 5, Gower's tract; 19, left restiform body; 20, right restiform body; 23, hypoglossal nucleus; 25, degeneration in left pyramid; 26, peri-olivary degeneration; 29, dorsal vagus nucleus; 30, root fibers of vagus nerve; 31, Deiters' nucleus; 32, descending vestibular root; 33, spinal root of fifth nerve; 34, median vestibular nucleus; 35, olivocerebellar fibers; 39, posterior longitudinal fasciculi.

hilum and those traversing the gray substance to attain the periphery were well preserved. The left olive was not without alteration. This was due to a marked fiber decrease in its outer half shown in the strands traversing the gray substance and evidently continuous with the peri-olivary degeneration presently to be described. The fine fiber felt-work between the cells in the gray substance was also much less marked than in the opposite or degenerated right olive. The gray matter,

however, was apparently well conserved. The cellular changes in the right medial and right dorsal accessory olives were similar to those found in the right inferior olive. The left olivocerebellar fibers were degenerated (Fig. 7). The left nucleus lateralis was, possibly, poorer in cells than the right. The left olive was brought out in relief by a degeneration of fibers lateral and ventral to it—peri-olivary fibers (Figs. 5 and 6, No. 26). A degeneration was traced cephalad as well as caudad in relation to the olive, and it seemed to be continuous. This will be discussed later. The median fillet was less darkly stained on

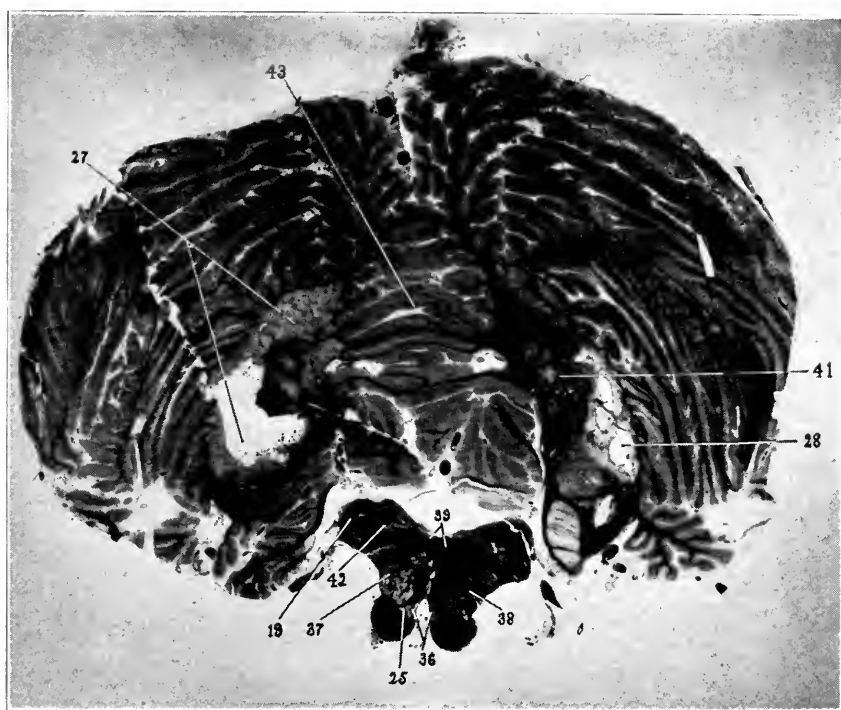


Fig. 8.—Transverse section of cerebellum and of medulla at emergence of glossopharyngeal nerve: 19, left restiform body; 25, degeneration in left pyramid; 27, softening in left cerebellar hemisphere; 28, softening in right cerebellar hemisphere; 36, arcuate nuclei; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 41, right dentate nucleus; 42, root fibers of glossopharyngeal nerve; 43, vermis.

the left side, and the crossing right internal arcuate fibers to it from the right posterior column nuclei were greatly diminished in number (Fig. 4, No. 12). The right nucleus cuneatus was poor in cells. The left anterior and anterolateral external arcuate fibers were degenerated (Figs. 3 and 4, No. 3).

In the left pyramid at its median and dorsal aspect there was a degeneration of fibers which was quite distinct in the region of the olive (Figs. 5, 6 and 7, No. 25) but it became much less so at the pyramidal decussation and was not seen in the cervical cord. Upward this degeneration was lost in the pyramidal bundles traversing the pons. In its greatest extent the degeneration seemed to radiate from the left arcuate nucleus. The arcuate nuclei, except in their lower portions, stained poorly with cochineal. There was no remarkable difference in their appearance.

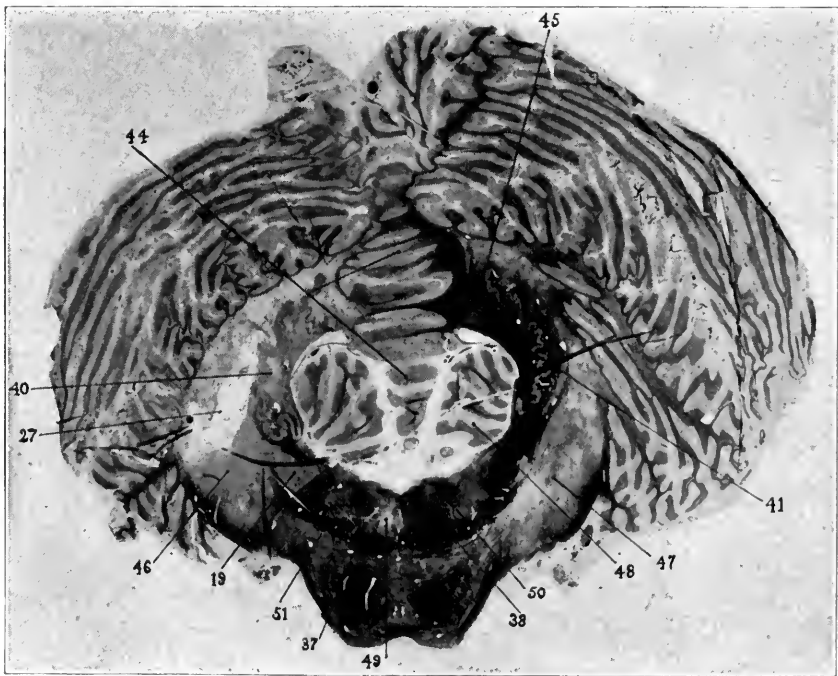


Fig. 9.—Transverse section of medulla and of pons through nucleus of sixth nerve: 19, left restiform body; 27, softening in left cerebellar hemisphere; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 40, degeneration of left dentate nucleus; 41, right dentate nucleus; 44, inferior vermis; 45, nucleus globosus; 46, left middle cerebellar peduncle; 47, right middle cerebellar peduncle; 48, tonsil; 49, vascular lesion in left median fillet; 50, abducens nucleus; 51, trapezoidal fibers.

The middle peduncles were severely degenerated on both sides (Figs. 9 and 10, Nos. 46 and 47), and the transverse fibers of the pons were degenerated (Fig. 11, No. 61). This was also true of the ascending pons fibers to the tegmentum. In contrast to the fiber degeneration, the pontile nuclei were remarkably conserved. In the tegmentum the left median fillet was seen to be involved by the small central lesion mentioned above (Fig. 9, 49). Some of the fibers of the

left posterior longitudinal bundle were involved. Also a degeneration of the left central tegmental tract was present over the normal left olive (Figs. 8, 9, 10 and 11, No. 37). The corpora quadrigemina appeared to be normal.

On the left side the cells of Deiter's nucleus might be somewhat diminished in number, as well as the fibers of the descending vestibular root (juxta restiform body. On the same side, in higher sections, con-

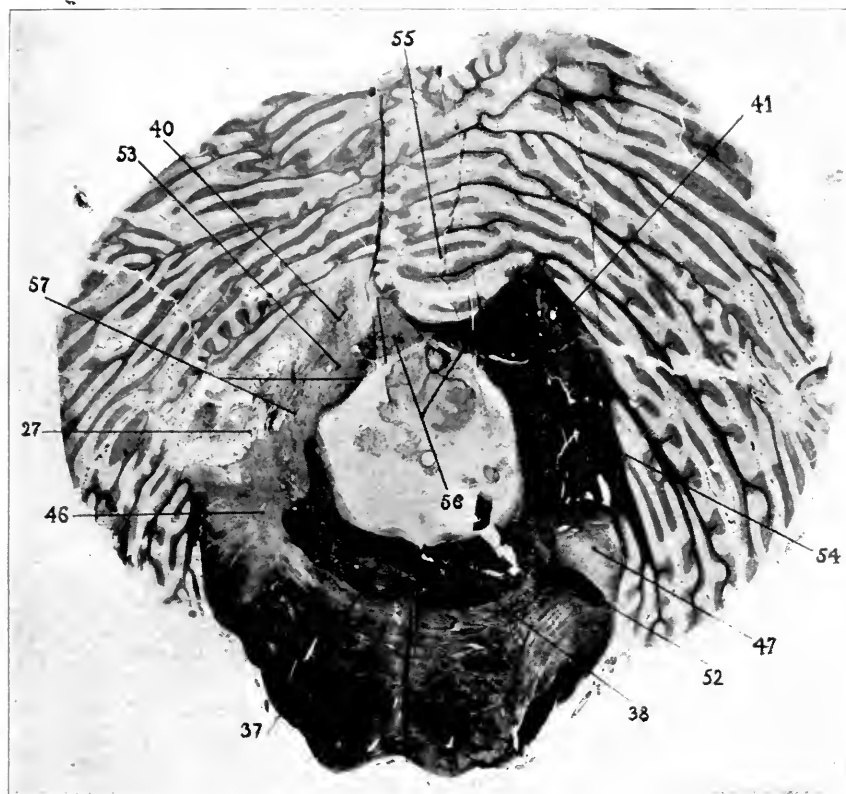


Fig. 10.—Transverse section of cerebellum and of pons at emergence of fifth nerve: 27, softening in left cerebellar hemisphere; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 40, degeneration of left dentate nucleus; 41, right dentate nucleus; 46, left middle cerebellar peduncle; 47, right middle cerebellar peduncle; 52, fibers of fifth nerve; 53, degeneration of left superior cerebellar peduncle; 54, right superior peduncle; 55, superior vermis; 56, roof nuclei; 57, fibers between tegmentum pontis and roof nuclei.

served strands were seen which extended between the tegmentum pontis and the roof nuclei (Fig. 10, No. 57). There was a lateral portion which lay lateral to the degenerated superior peduncle and a medial por-

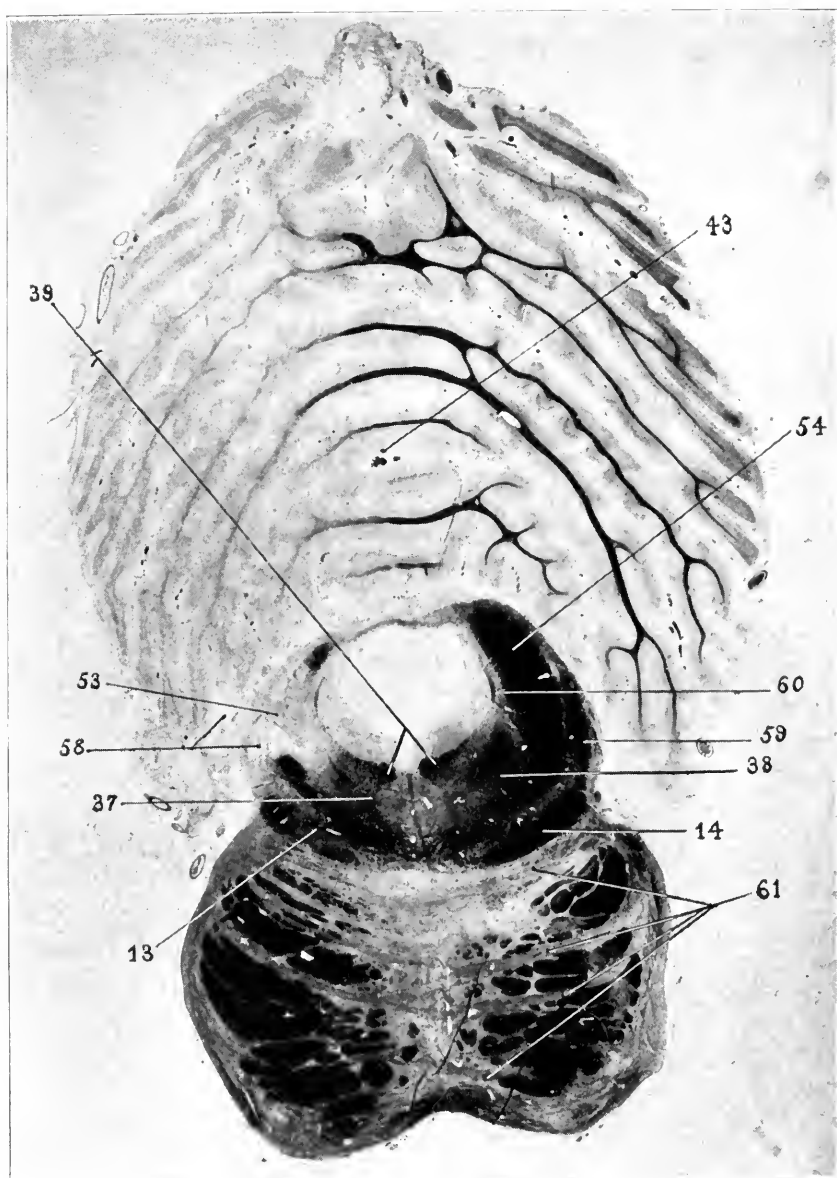


Fig. 11.—Transverse section through the isthmus: 13, degeneration of left median fillet; 14, right median fillet; 37, degeneration of left tegmental tract; 38, right central tegmental tract; 39, posterior longitudinal fasciculi; 43, vermis; 53, degeneration of left cerebellar peduncle; 54, right cerebellar peduncle; 58, vascular lesion involving left lateral fillet; 59, right lateral fillet; 60, mesencephalic root of trigeminal; 61, degeneration of transverse pontile fibers.

tion bordering the ventricular wall. Fibers in the decussation of the roof nuclei were continuous with the foregoing in a sweeping curve. It is probable that these fibers represented the nucleocerebellar tract or the fastigiobulbar tract, or both. The right roof nucleus was intact, the left relatively little less poor in number of cells or staining qualities.

Because of the changed direction of the cuts from transverse to horizontal in the second block, which included the peduncular and subthalamic regions, some of the lower sections were incomplete. Also, in the reconstruction of the specimens, the right half of these sections was misplaced posteriorly and slightly downward.



Fig. 12.—Horizontal section through red nucleus. The right half of the section and following sections are misplaced posteriorly and slightly downward because of the reconstruction of the specimen: 14, right median fillet; 37, degeneration of left central tegmental tract; 38, right central tegmental tract; 39, posterior longitudinal fasciculus and nucleus of third nerve; 62, degenerated right red nucleus; 63, root fibers of oculomotor nerve; 64, inferior colliculus; 66, substantia nigra; 67, pes pedunculi; 68, vascular lesion in right pes pedunculi; 69, optic tract; 70, hippocampus; 71, inferior longitudinal fasciculus.

The right red nucleus (Figs. 12 and 13, No. 62) showed a marked degeneration as compared with the left side, shown by the poverty of transversely cut fibers. Above the degenerated red nucleus in the subthalamic region (Fig. 14, No. 78) there was a considerable thinning of fibers in Forel's field, notably in the thalamic bundle of Forel (Déjerine) (Figs. 15 and 16, No. 88). Yet these fibers seemed to be distinct from a bundle which was in relation to the mammillothal-

amic tract, and also designed by this author as part of the thalamic bundle. The lenticular bundle of Forel, *ansa lenticularis*, and inferior thalamic peduncle were unaffected (Fig. 15). In the optic thalamus, neither in the external nor internal medullary laminae or in the external nucleus of this body, could degenerations actually be demonstrated. For this reason, sections of the basal ganglia are not shown in the illustrations (Block 3). It is quite remarkable that the demonstrable degenerations above the red nucleus were so slight. This would suggest considerable autonomy of the cerebellum and this nucleus.

In sections below the olive, replacing the peri-olivary degeneration on the left, there was a degeneration of fibers extending caudalward



Fig. 13.—Horizontal section through optic tract: 62, degenerated right red nucleus; 64, inferior colliculus; 67, pes pedunculi; 69, optic tract; 70, hippocampus; 71, inferior longitudinal fasciculus; 72, inferior brachium; 73, superior brachium; 74, anterior pillar of fornix; 75, external geniculate body.

in the anterolateral bundle of fibers to the cord in the angle between the pyramid and the afferent cerebellar cord tracts (Fig. 3, No. 1). In the upper cervical cord it was traced as a triangular degeneration with its base at the periphery in the lateral column (Fig. 2); in the middle cervical region (Fig. 1) it was very much less marked and superficial, and it could not be found in the cervical enlargement. This degenerated fiber bundle was without doubt Helweg's tract. In the anterior column on the opposite or right side there was a slight thinning of fibers lateral to the direct pyramidal tract (Figs. 1 and 2 A).

It could be traced through the cervical cord and even in diminishing volume in the dorsal cord, but could not be seen in the lumbar region. This degeneration probably marked the descending cerebellospinal tract. The spinocerebellar tracts were not degenerated.

INTERPRETATION AND DISCUSSION

The foregoing is the report of a case in which there is a destruction of tissue in both cerebellar hemispheres with conservation of the vermis and the central vestibular system, and whose predominant symptomatology is marked dyssynergia, cerebellar catalepsy and scanning speech. Equally important from the standpoint of negative symptomatology is



Fig. 14.—Subthalamic region. Horizontal section through superior colliculus; 65, superior colliculus; 69, optic tract; 73, superior brachium; 74, anterior pillar of fornix; 75, external geniculate body; 76, internal geniculate body; 77, pulvinar; 78, degeneration in right Forel's field; 79, mammillothalamic tract (Vieq d'Azyr); 80, lenticular nucleus; 81, Luys' body; 82, posterior commissure.

the absence of spontaneous nystagmus and of spontaneous errors of pointing after Bárány.

Cerebellar catalepsy was first described by Babinski¹ in a patient who presented, as in the case reported, a dissociation of the two kinds of volitional equilibrium: asynergia and catalepsy. He considered the latter as a symptom of cerebellar disease and, in discussing the underlying pathology quotes Dupré and Devaux, and Léopold Lévi as observing similar states in cerebellar abscess. In a later communica-

tion² Borgherini and Gallerani are quoted as observing a similar condition in animal experimentation on the cerebellum; and Rossi, in a case of parenchymatous atrophy of the cerebellum, noticed that this symptom was quite marked. Recently, La Salle Archambault³ observed a similar case in which the degeneration of the Purkinje cells was the predominating and only constant feature. The central nuclei, olivary bodies and olivary cerebellar system, showed only insignificant secondary retrograde atrophies.

Déjerine⁴ has stated that this symptom lacks anatomic confirmation as being due exclusively to a cerebellar lesion. Oppenheim⁵ considered

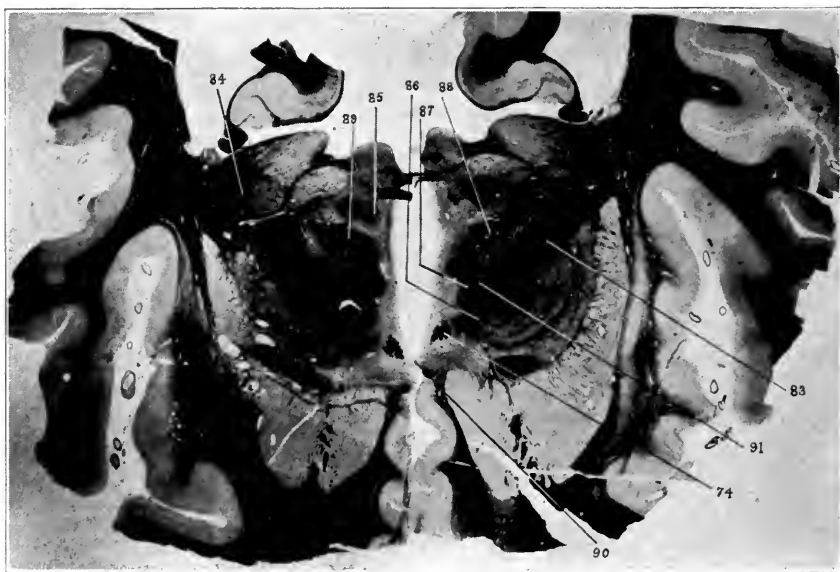


Fig. 15.—Subthalamic region. Horizontal section superior to Figure 13: 74, anterior pillar of fornix; 83, internal capsule; 84, Wernicke's field; 85, fasciculus retroflexus (Meynert); 86, ansa lenticularis; 87, inferior peduncle of thalamus; 88, degeneration in right thalamic bundle of Forel; 89, left thalamic bundle of Forel; 90, posterior commissure; 91, lenticular bundle of Forel.

this symptom quite rare and had not encountered it in his experience up to the year 1913.

The absence of nystagmus is explained by the practically intact vestibular system. This case confirms the opinion of Wilson and Pike⁶

3. Archambault, LaSalle: Parenchymatous Atrophy of the Cerebellum, *J. Nerv. & Ment. Dis.* **48**:273, 1918.

4. Déjerine: *Sémiologie System Nerveux* **1**:424, 1914.

5. Oppenheim: *Lehrbuch der Nervenkrankheiten* **2**:1380, 1913.

6. Wilson, J. G., and Pike, F. H.: The Differential Diagnosis of Lesions of the Labyrinth and of the Cerebellum, *J. A. M. A.* **65**:2156 (Dec. 18) 1915.

that rhythmic nystagmus or labyrinthine nystagmus consisting of slow and quick components is properly a symptom of vestibular disease. They distinguish between this form and cerebellar nystagmus, characterizing the latter as an asynergia of ocular muscles of oscillatory nature. In Archambault's recent case of cerebellar atrophy with intact vestibular system, he speaks of "nystagmic shocks" rather than true nystagmus interpreted either as a tremor of the ocular muscles or perhaps as a manifestation of asynergy. Oppenheim, admitting that nystagmus is frequently given in the symptomatology of cerebellar lesions, doubts



Fig. 16.—Subthalamic region. Horizontal section superior to Figure 14, passing through semilunar nucleus: 65, superior colliculus; 74, anterior pillar of fornix; 75, external geniculate body; 76, internal geniculate body; 77, pulvinar; 79, mammillothalamic tract (Vicq d'Azyr); 80, lenticular nucleus; 84, Wernicke's field; 88, degeneration in right thalamic bundle of Forel; 89, left thalamic bundle of Forel; 92, semilunar nucleus of Flechsig; 93, anterior segment of internal capsule; 94, caudate nucleus.

whether it may be due directly to cerebellar lesions. In 1915, I expressed the opinion that nystagmus properly speaking is not a symptom of disturbed cerebellar function.

The normal outcome of pointing tests, although incomplete and only tested by spontaneous pointing and not after turning or syringing, are

significant. From the standpoint of cerebellar function, the discussion relevant to the above may be best taken verbatim from Jones' book on "Equilibrium and Vertigo," page 189:

Summarizing, therefore, the cerebellum plays only a partial rôle in the large mechanism of the pointing. The kinetic-static sense, the arthroidal sense, tactile, auditory and visual impressions and memory all combine to inform the individual of the position of an external object. The motor areas of the cerebral cortex then send impulses to the arms; the function of the cerebellum is merely in controlling the execution of the cerebral mandate. If this be erroneous, because of the vertigo, past-pointing results.

The symptoms of dyssynergia, dysmetria and dysdiadokokinesia were marked and characteristic. They would tend to confirm our prevalent notions regarding cerebellar symptomatology. In contrast to the above symptoms was the conservation of trunkal static equilibrium as evidenced by the ease with which this patient maintained the sitting position. Mills and Weisenburg⁸ insist that when trunkal movements are affected the vermis must be involved in whole or in part. The intact vermis and its related roof nuclei in the present case are again emphasized in this connection. Cerebellar catalepsy in this case is another example of conserved static equilibrium contrasted with disturbed dynamic equilibrium exemplified by the great incoordination in the extremities.

From the anatomic standpoint, the principal findings are corroborative in general of the degenerations following cerebellar defects such as are demonstrated in cases of cerebellar lesions, animal experimentation, and particularly unilateral and bilateral agenesis of the cerebellar hemispheres and reported by Edinger,⁹ Anton and Zingerle,¹⁰ and Oliver Strong.¹¹ But as emphasized by Anton, the degenerative changes following congenital defects are not necessarily identical with degenerations following later lesions because of the involvement of associated neurons in the former. Worthy of mention, and perhaps throwing some light on the anatomic connections of the cerebellum and of the inferior olive, are the following considerations:

1. The destruction of the left restiform body may be brought into relationship with the destruction of the left dentate nucleus for the reason that the right restiform body is practically intact with an extensive destruction of both cerebellar hemispheres and conservation of the right dentate nucleus.

8. Mills, C. K., and Weisenburg, T. H.: Cerebellar Symptoms and Cerebellar Localization. *J. A. M. A.* **63**:1813 (Nov. 21) 1914.

9. Neuberger and Edinger: *Berl. klin. Wchnschr.* **35**:69, 1898.

10. Anton and Zingerle: *Genaue Beschreibung eines Falles von beiderseitigem Kleinhirnmangel*, *Arch. f. Psychiat.* **54**:8, 1914.

11. Strong, Oliver: A Case of Unilateral Cerebellar Agenesis, *J. Comp. Anatomy* **25**:361, 1915.

2. On the left side the degenerations in the central tegmental tract, circumolivary fibers, internal fibers of the olive, and Helweg's bundle seem to indicate a more or less intimate connection of these structures. Helweg's original anatomic studies¹² by means of carmin sections led him to the conclusion that his wedge-shaped spinal bundle was continuous above with the fibers surrounding the inferior olive and cephalad with his "oval" bundle, which is doubtless the tract now known as the central tegmental tract. Helweg believed that the fibers of the oval bundle formed the posterior commissure. The upward continuation of the degenerated central tegmental tract could not be traced in our case above the red nucleus. In the reported cases of agenesis above mentioned, there is no uniformity in the degeneration of these structures. In Edinger's case there was an absence of the right cerebellar hemisphere. The right central tegmental tract above the normal right olive was markedly defective. The degeneration was traced to below the anterior quadrigemina. Helweg's bundle was not mentioned. In the bilateral agenesis of Anton and Zingerle the central tegmental tract was not degenerated, but there was a bilateral degeneration of both Helweg's tracts. These authors draw a close relationship between these degenerations and the degenerated inferior olives. In Oliver Strong's case, in which the left cerebellar hemisphere was missing, the right central tegmental tract was degenerated, Helweg's bundle not being mentioned.

3. On account of the small tegmental lesion, which is in close vicinity to the central tegmental tract, the question must remain an open one as to whether this tegmento-olivo-spinal degeneration is dependent on it or on the cerebellar lesions. In a careful study of the serial sections representing the tegmental lesion no actual defect in the bundle can be demonstrated due to softening. This lesion probably explains the degeneration of the left median fillet which it directly involves. Regarding the significance of the above tegmental lesion and the other two small lesions in this case, we do not believe that they affect to any considerable degree the clinico-pathologic interpretations.

12. Helweg: Studien über den centralen Verluft der Vasomotorischen Nervenbahnen 19:104, 1887.

A CASE OF ACROMEGALIA ASSOCIATED WITH BRAIN TUMOR

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INTRODUCTION

The principal interest in this case is the relation between the cerebral tumor and the acromegalic symptoms. To prove which of these was primary or causative is impossible, but we will endeavor to show that the entire picture can be explained on the assumption that the tumor was the original lesion and the polyglandular symptoms were dependent on pressure with resultant biochemical changes. There is abundant support in the literature for this contention.

This patient has been under observation at various hospitals since March, 1914, with the diagnosis of acromegalia, the tumor mass not being suspected.

CLINICAL OBSERVATIONS

History.—J. B., Case 18457; Necropsy 2072. The patient was admitted to the Danvers State Hospital, Feb. 15, 1915. The family history was negative for nervous and mental diseases. The patient was born in 1865. Birth and early development were uneventful. He was married in 1890 and had six children. His wife had had no miscarriages. At the time his illness began, he was a foreman in a shop. He had no venereal disease. He used alcohol and tobacco moderately. His trouble began about 1895. The first symptoms were a sense of pressure and discomfort in the head with enlargement of the head, hands and feet, bowing of both legs and loss in weight. Progress was gradual, and the patient continued to work as a machinist. In the spring of 1903 he sustained a so-called shock. This caused paralysis of the left arm and leg and incapacitated him for about three months. In 1910 he began to have severe, almost constant headaches. At times they were diffuse but usually they were occipital. The pain in the hands became more acute. He remained at work until 1913 when he was finally compelled to resign. He became irritable and slept poorly.

He was admitted to the Psychopathic Department of the Boston State Hospital, March 19, 1914. Here he was cooperative and showed considerable insight, good orientation, very fair memory and no delusions or hallucinations. He was depressed and contemplated suicide because of his apparently hopeless condition.

Examination.—There was thickening of the cutaneous tissues over the supra-orbital ridges, fingers, hands, toes, feet and nose. The hands were markedly enlarged and the tongue thick. The heart showed a systolic murmur. Exophthalmos was present, pupils and fundi were normal. There was a positive von Graefe sign. The left knee jerk was sluggish, the right was obtained only with reinforcement. Other reflexes were sluggish but equal. The patient had a waddling gait. Cutaneous sensibility and special senses

were unimpaired. The blood pressure was systolic, 140; pulse, 105; respiration, 20. The left lobe of the thyroid gland was enlarged. The Wassermann reaction with blood was negative; the spinal fluid was normal. Polydipsia and polyuria with vomiting had been persistent for six months before admission. The sugar content was 8 per cent.

Treatment and Course.—On April 18, 1914, a transsphenoidal operation was attempted. A large sella turcica was exposed and the bulging dura incised. Every attempt to excise the exposed gland led to such an amount of hemorrhage that no tissue was removed. On May 2 the wound was reopened and a radium tube inserted into the gland substance. The tube was left in situ for six hours. There was complete subsidence of his headache and the patient was discharged May 16.

Visual acuity was 20/15 in either eye, and there was only 0.3 per cent. sugar. The neuralgia on the right side had entirely disappeared, and the patient was free from headache and vomiting.

He returned to work until Thanksgiving, 1914, when he quit, principally on account of mental symptoms. He became more irritable, lost interest in his work, showed considerable memory loss, was drowsy, developed ideas of poisoning and thought his wife and daughter were persecuting him. He became violent at times and used obscene language. He threatened to commit suicide.

On admittance he was quiet in manner and cooperative. At times he became excited, spoke loudly and forcibly. There was no speech disturbance. For a few hours he was not clearly oriented. School knowledge and calculations were fairly accurate. His handwriting was normal. Memory was excellent. The patient talked clearly but at times showed some confusion. Apparently he had delusions of persecution; he complained of poison being placed in his food. He had no insight. He showed considerable irritability and at times was somewhat impulsive and hard to manage.

He was a moderately well nourished man. His nose was much enlarged and twisted to the right. The inferior maxilla was prominent. His hands were much enlarged, especially around the joints. His feet were enormous in size, the legs much bowed. He had hypertrophy of the tibia and femur, enlargement of the knee, wrist and metatarsophalangeal joints. There was increased fremitus at the right apex and a systolic murmur was transmitted to the axilla. There was marked sclerosis of the peripheral arteries. The systolic pressure was 155. The tongue was large and red. Urine: The specific gravity was 1.026; sugar, 0.4 per cent. The blood and spinal fluid Wassermann reactions were negative. There was a slight increase in globulin and albumin but the colloidal gold test was negative.

The left pupil was larger than the right; both pupils were moderately dilated and reacted fairly well to light and accommodation. Vision of the right eye was 20/40, of the left 20/50. The fundi showed a thickening of the retinal vessels and some evidence of choked disk in both eyes. Cutaneous sensibility and special senses were normal. Deep reflexes were absent; superficial reflexes were present and equal. There was a marked Romberg sign and considerable tremor of the hands.

Head Measurements: The head measurements were: biparietal, 15 cm.; occipitofrontal, 20 cm.; mentobregmatic, 27 cm.; occipitomenal, 22 cm.; bitemporal, 11 cm.; intermastoid, 14 cm.; interzygomatic, 14.5 cm., and the cephalic index 75 cm.

The patient was up and dressed shortly after admittance. He was sent to the arts and crafts department where he became much interested in copper

work. He was pleasant, agreeable and cooperative. He complained of pain in the legs, a vague feeling of discomfort in his abdomen, and occasional headaches, diffuse in character.

He continued in this state until August, 1916, when he began to complain of increased dizziness which caused him to remain in bed, at first only a portion of the time, but later continuously. He became more unsteady in his gait and was unable to get about alone. Pain in the head increased, being more marked over the vertex. There was considerable loss of weight and strength. The left side was much weaker than the right. The deep reflexes on the right were greater. Hypertrophy of the face and joints and

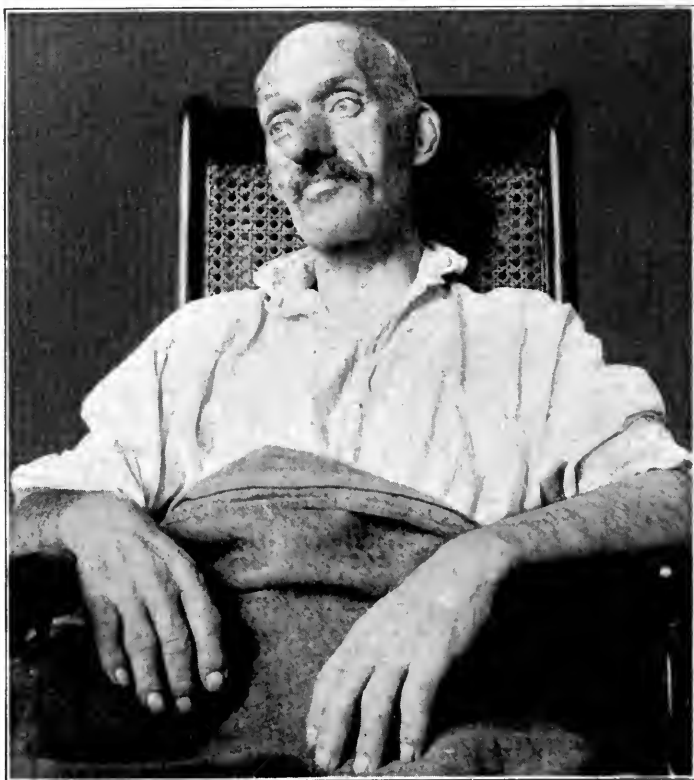


Fig. 1.—Patient seven months before death, showing characteristic changes of acromegaly.

bowing of the legs had increased. He was clumsy and awkward and there was considerable impediment in speech. Sensation was normal but hearing was better on the left. Optic atrophy was more pronounced on the right. He showed a rather childish pride in his condition with occasional attacks of irritability, but apparently the ideas of persecution had disappeared.

He continued to fail mentally and physically until he died, April 26, 1918, apparently of bronchopneumonia.

Postmortem Observations: Gross Anatomy.—Postmortem rigidity was present to some extent. The pupils were equal and regular, 4.5 mm. in

diameter. The ciliary vessels at the junction between iris and cornea were greatly injected on the right. The left eye was more prominent than the right. The nose measured 8.5 cm. in length and at the lower portion was 4.2 cm. in width. The supra-orbital crest was massive. The circumference of the head was 58 cm.; the distance from the top of the head to the mandible was 87.5 cm. The chin was prominent and greatly thickened. The transverse diameter of the head was 14 cm., the antero-posterior diameter 20 cm. The zygomatic processes and superior maxillary bones were greatly enlarged, the lips unusually thick. The forearms appeared slightly curved. The right wrist was 18.5 cm. in circumference, the left 18.7 cm. The terminal phalanges were greatly flattened. The measurements of the hands were: right hand: circumference, 23.8 cm.; index finger, length, 11.5 cm.; circumference middle phalanx, 8 cm.; circumference terminal phalanx, 7 cm.; middle finger, length, 11.5 cm.; circumference middle phalanx, 8.1 cm.; circumference terminal phalanx, 6.8 cm.; left hand: circumference, 22.5 cm.; index finger, length, 10.7 cm.; circumference middle phalanx, 7.5 cm.; and circumference terminal phalanx, 6.5 cm.

With heels together, the distance between the knees was 16.5 cm. There was marked bowing of the tibiae and the tibial crests were slightly roughened and flat, especially the left. The right ankle was 23 cm. in circumference, the left 23.5 cm. The right foot was 24.5 cm. long, the circumference at the ball of the foot 23.5 cm. The left foot was 24 cm. long, the circumference at the ball 24.5 cm. The toes were greatly enlarged and flat. There was exostosis at the joints. There was a heavy, hairy growth over the arms, pubis and legs; no adenopathy. The skin over the chest was very thick; there was practically no fat. Over the abdomen the fat measured 1 cm.

Head: Measurements: frontal, 1 cm.; temporal, 0.3 cm.; occipital, 0.8 cm. Above the interior occipital protuberance and to the right, there was an irregular, worm eaten erosion into the diploic space. The scalp seemed thinner than the external measurements indicated. The dura was adherent. The frontal sinuses were tremendously enlarged. The dura over the right occipital and parietal lobes had been destroyed and an irregular nodular hemorrhagic area was seen over the hemisphere along this side. The pituitary was rather firmly adherent to the sella turcica. The sella measured 2.5 cm. by 2.5 cm., and was 1.5 cm. deep. On either side of the sella floor was a depression into which the fifth finger fitted snugly. In the thin posterior wall of the sphenoid sinus was a round hole 0.6 cm. in diameter, which communicated with the nasal cavity.

Brain: Over the right parietal and occipital region there was a large, nodular, irregular tumor mass which was easily removed from the brain. It measured 8.5 by 9.5 cm. and consisted of about fifteen nodules, the smallest measuring about 1 cm. in diameter and the largest 7 cm. Some of them were darkly pigmented, others were white and glistening. The upper surfaces were firmly attached to the dura while the lower surface was smooth and covered by a thin layer of fibrous membrane. Capillaries in the pia were greatly injected. There was no sclerosis of the basal vessels. The olfactory bulbs were large but not adherent.

The whole parietal region was pressed down by the tumor mass which was separated by the pia mater from the brain substance. The tumor was not, therefore, derived from the brain substance but from the meninges. The markings of the convolutions in this depressed area had entirely disappeared and were not visible. The depressed convolutions were the upper part of

the anterior and posterior central convolutions, the paracentral lobule, superior parietal lobe and precuneus. The lower part of the precuneus, the posterior part of the cingular gyrus, the anterior part of the lingular gyrus, and the posterior part of the hippocampal gyrus were pressed out behind the splenium of the corpus callosum. The posterior two thirds of the cingular gyrus were pressed out over the corpus callosum, compressing the corresponding part of the left hemisphere. The third ventricle was thus pressed between the tip of the temporal lobe (which was also markedly compressed and flattened) and the posterior two thirds of the cingular gyrus. The occipital lobe was com-

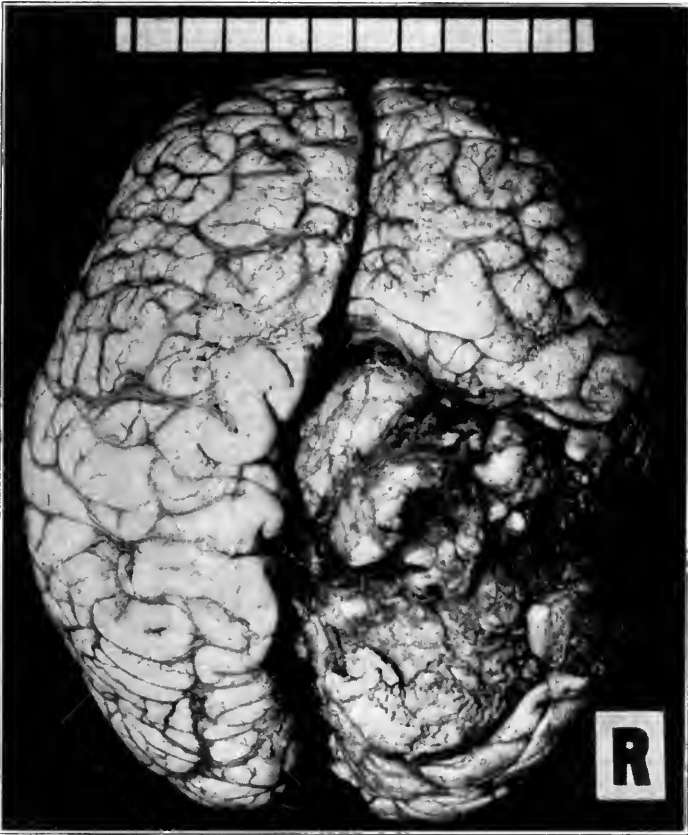


Fig. 2.—Superior surface of the brain showing tumor masses and flattening of convolutions.

pressed from the origin of the calcarine fissure to the tip of the occipital lobe, measuring 2.9 cm., while the left side measured 3.8 cm. The left hemisphere showed general flattening of the convolutions. The middle part of the median aspect of the left hemisphere showed marked concavity.

The corpus callosum was pressed down and the middle part appeared somewhat thinner. In consequence of pressure on the corpus callosum, the septum lucidum was folded up into three parallel wrinkles. The interventricular foramen was open. The body of the fornix was flattened. The intermediate

mass of the thalamus appeared flattened. The optic recess was somewhat distended, especially on the right side, and the optic nerves were markedly flattened on both sides. The infundibulum was pressed down. The pineal body and pineal recess seemed slightly compressed. The third ventricle was thus generally much flattened. Pressure of the tumor on the third ventricle seemed especially strong in the middle part, i. e., on the pituitary body.

From the details of the complete necropsy examination may be noted: Other findings corresponded to the clinical examination; they were negative

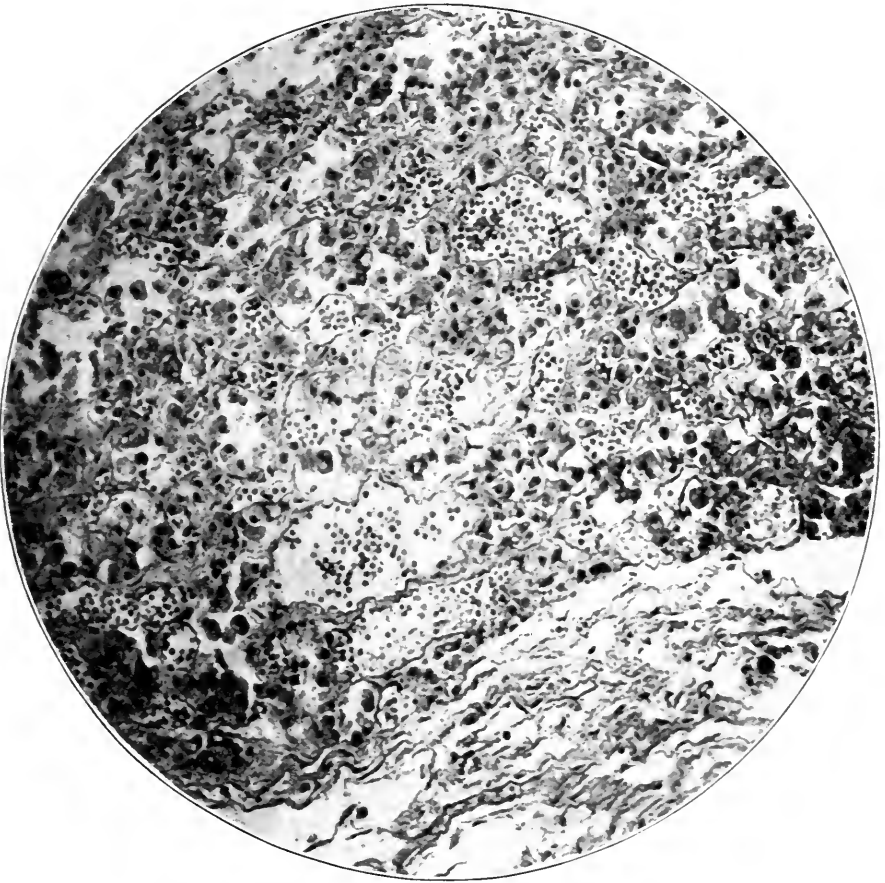


Fig. 3.—Histologic structure of the marginal zone of the pituitary body.

or unimportant. There were increase of pericardial fluid and chronic endocarditis; the coronaries were slightly sclerotic. The aorta was thickened and sclerotic. The liver showed a tendency to nutmeg appearance and fatty streaks. Chronic diffuse nephritis was present. There were yellowish spots in the cortex of left suprarenal, which was smaller than the right: the medulla of the right was thicker and there was a hemorrhagic spot beneath the capsule. The left lobe of the thyroid gland was nodular and hard and weighed 45 gm.; the right weighed 15 gm. The pons, midbrain and cord were soft.

Histologic Examination.—Heart: Fibers somewhat atrophic. Some fatty infiltration seen beneath epicardium. Aorta: Slight sclerosis. Lungs: Early bronchopneumonia; marked congestion. Spleen: Acute splenic tumor, hyperemic. Liver: Chronic passive congestion; central atrophy and necrosis; periportal increase of connective tissue, with lymphocytic infiltration. Pancreas: Marked chronic pancreatitis; sclerosis of many Isles of Langan; fatty infiltration of some lobules seen. Kidneys: Cloudy swelling and marked congestion. Testicles: Paucity of interstitial tissue with few cells of Leidig;



Fig. 4.—Chromophobe struma, showing both marginal (relatively normal) and central zone.

parenchyma slightly atrophic. Prostate: Lymphocytic infiltration and some glands showed definite tendency to adenomatous transformation. Thyroid: Left lobe, marked increase of interstitial tissue and hyperplasia of epithelial cells with practically no colloid. Right, marked hyperplasia of parenchymatous elements with excess of colloid; tendency to interstitial proliferation. Suprarenals: Fatty degeneration and lymphocytic infiltration in zona fasciculata of the cortex. In the cortex were areas of adenomatous transformation. The medullar cells were pigmented.

Pituitary Body: The dural capsule was remarkably thickened. Most of the posterior lobe showed degeneration and was torn away when taken out. The anterior lobe was hyperplastic in its entire body except in a narrow marginal zone. This marginal zone presented still sinusoidal structure with luxuriant vascularity. Almost all cells were neutrophilic. The rest of the pituitary consisted of a more or less loosely packed body of epithelial elements with no demonstrable connective tissue and practically no blood vessels. Toward the center of the lobe the cell elements were arranged more

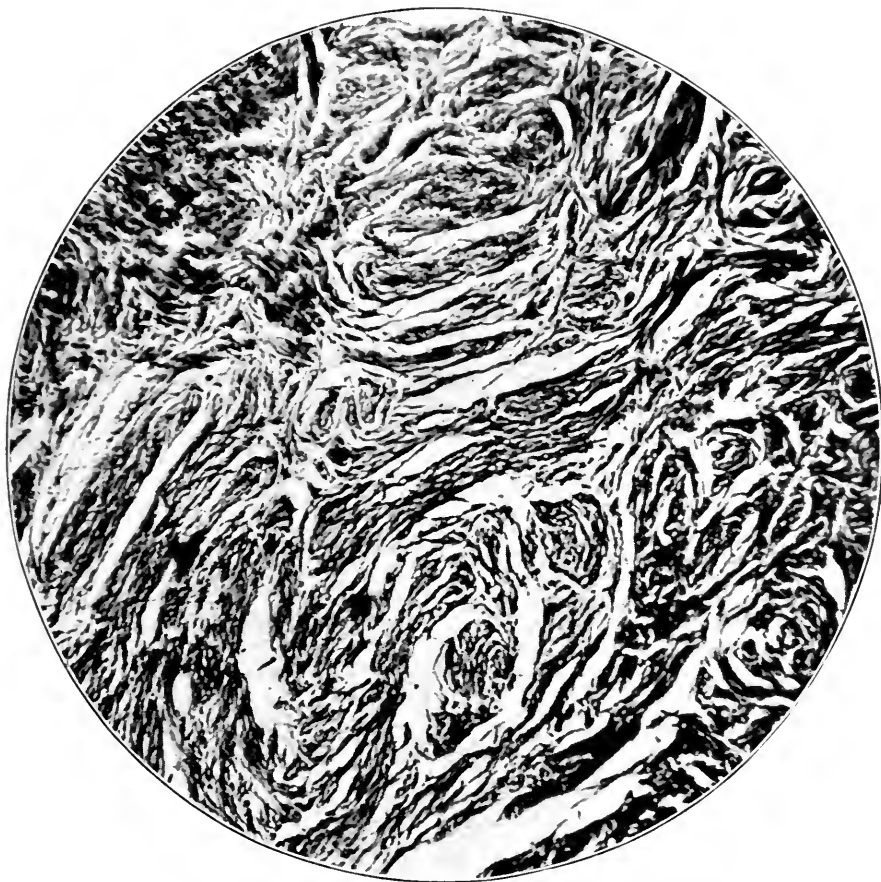


Fig. 5.—Histologic picture of the endothelioma.

irregularly and were widely separated. Except in the marginal zone no sinusoidal or alveolar structure was discerned. In the center of the gland practically all cell elements were neutrophilic. In a part of the hypertrophic central region there was evidence of increased secretion of colloidal matter, the perivascular spaces being filled with a pinkish stained homogeneous matter. No mitotic figures were observed.

The condition of the pituitary body was no doubt pathologic, exhibiting adenomatous characteristics. It corresponded with the chromophobe struma of Cushing's nomenclature and also with the malignant adenoma of early writers.

Histology of the Tumor.—The tumor was made up of long endothelial cells with elongated nuclei. They were rather regularly arranged showing a parallel course and a concentric arrangement around the vessels. The vessels were rather scant. Although the cells were mostly elongated, there were a number of different forms. Some were round, the nuclei being fat and regular in shape. They were not arranged regularly, but showed a bizarre order. The nuclei of the tumor cells showed also a remarkable polymorphous condition. Some



Fig. 6.—Cut surface of the brain according to Dalton's method.

were round and small, others oval, spindle, rod shaped and much elongated. A great number of nuclei showed direct division, while many showed shrinkage, vacuole formation, etc. Apparently there had been both a proliferating and a regressive process going on simultaneously, with the former progressing more slowly.

Observation of Cut Surface of the Brain According to Dalton's Method.—The herniation of the right gyrus fornicatus was well observed in the cut sur-

faces. The right temporal lobe was much compressed and flattened. The right lateral ventricle was practically obliterated. The left ventricle was also remarkably compressed in its middle part where it was seen only as a narrow crevice.

COMMENT

The symptoms of this patient may be divided into those of acromegaly, exophthalmic goiter, cerebral tumor and mental manifestations due to organic lesion.

Acromegalic Symptoms.—Marie represents the view which attributes acromegaly to diminished function of the pituitary body. Strümpell, Guerrini, Cagnett and others believe that this disease is due to nutritional disturbances, the enlargement of the pituitary being only secondary. The hypertrophy of the gland is regarded by others as even accidental. Massalongo, Tamburini, Benda, Modena, Fisher and Cushing favor the hyperpituitarism conception because of the usual finding of glandular hypertrophy, the opposite clinical manifestation from experimental extirpation of the anterior lobe, the low carbohydrate assimilation in the earlier stage of the disease, improvement of the disease from a partial extirpation of the hyperplastic gland, etc. A great many authors have observed hypertrophic enlargement of the gland and a histologically demonstrable hyperplasia, and it is probable that the latter view is correct.

In the present case, as in most others, a condition which was called by Cushing "chromophobe struma" was found; also evidences of hypersecretion.

The adenomatous transformation of the gland in this case, however, is not to be considered as "malignant" in the sense used by earlier observers. We regard it merely as an extreme hyperplastic condition of the gland, for these reasons:

1. In spite of the long standing disease the pituitary was only slightly enlarged and retained its original form.
2. The capsule was thickened and showed no tendency to rupture by the proliferated gland substance.
3. The marginal zone remained practically unaltered.

Cushing seems to hold the same opinion with regard to the nature of the hyperplasia. "It would appear," he says, "that any of these functionally unstable glands may, under certain biochemical stimuli, assume adenomatous characteristics, and doubtless most individuals afflicted with acromegaly face the possibility of such a transformation." We have in the present case, a definite histopathologic basis to account for the cynical syndrome, and we believe that this condition was responsible for the various symptoms.

Although it was impossible to examine the posterior lobe on account of the softening, it is obvious that this had nothing to do with the acromegalic syndrome. The syndrome of adiposity, high sugar tolerance, subnormal temperature, slow pulse, asthenia, etc., is accounted for by posterior lobe deficiency. The present case did not show any of these symptoms. The degeneration of the posterior lobe must, therefore, have been of late occurrence.

Exophthalmic Goiter.—In no one of Cushing's forty-seven cases was there symptomatic evidence of hyperthyroidism. Cushing admits that some degree of exophthalmos is common in hypophysial disorders, but he attributes this to the neighborhood effect of the glandular tumor. In our case the patient showed not only exophthalmos, but also struma, von Graefe's sign, tachycardia, etc., and they are, in all probability, to be regarded as evidence of hyperthyroidism. Moreover, the pituitary body was only slightly enlarged and no neighborhood symptoms were noted.

The thyroid was enlarged and asymmetrical. Microscopically, parenchymatous hypertrophy with luxurious vascularization, was demonstrated. There was also increase in the interstitial tissue of both lobes. The findings in the thyroid correspond to those of a typical case of exophthalmic goiter of long standing. The findings in the thyroid were entirely different from those of myxedema, although there was remarkable increase in the interstitial tissue. The latter shows an increase in old cases of exophthalmic goiter.

Cushing and others have reported cases with a polyglandular syndrome in which the symptoms of hyperadrenalism and hypo-adrenalism were apparent. It is quite possible, therefore, for the thyroid to show secondary symptomatic manifestations as a result of a lesion in the pituitary body.

Cerebral Tumor.—Microscopic examination showed the tumor mass to be typical endotheliomas. This tumor is of slow growth. Cushing describes two cases in which the lesion had been causing local symptoms for thirteen and nine years, respectively, before general pressure symptoms appeared. Most of the symptoms of brain tumor were observed in this case. About eight years after the onset of the disease, the patient had a so-called shock followed by paresis of the left arm and leg. This is to be regarded as a focal manifestation of the tumor. Four years before his death choked disk was discovered. During the last four years he suffered from constant headaches and periodical vomiting.

No doubt the diagnosis is clouded by the symptoms of acromegaly. However, if both general and focal manifestations had been thor-

oughly studied, it might have been possible to make a correct diagnosis, at least in the later stages of the disease.

Mental Symptoms.—Irritability, distrust, indecisiveness and lack of concentration are attributable to the hypophyseal derangement. Lack of interest, drowsiness and deterioration are probably due to increased intracranial pressure. The depression in the early stage of the disease, on the other hand, might have been nothing but physiologic reaction to his hopeless condition.

The Cerebral Tumor and Pituitary Body Disorder.—In the literature are a great many cases of acromegaly with cerebral tumor situated in the neighborhood or at a distance. Are the tumors found in these cases accidental, having nothing to do with the disturbances of the pituitary body? Cushing seems to be inclined to believe in the etiologic significance of these cerebral tumors in the development of hyperpituitarism and hypopituitarism, stating: "In every case of increased intracranial tension, from whatever source, there probably occur secondary changes in the hypophysis, often with gross deformations and resultant functional disturbances which frequently elicit recognizable clinical manifestations." Cushing observed a case of outspoken acromegaly with an unsuspected cerebellar cyst. "Whether the hypophyseal hyperplasia" he says, "was merely a concomitant process which bore no relation to the obstructive hydrocephalus, or whether the gland had been aroused into its state of pathologic overactivity as a secondary result of the cerebellar lesion cannot be positively certified. I incline toward the latter view."

In our case the third ventricle was not especially dilated, but the pressure of the tumor had been exercised directly on the hypophyseal fossa. Apparently the endothelioma dated back before the symptoms of acromegaly appeared. Whether or not the tumor had been playing a causative rôle is difficult to determine. However, it seems to us that it is quite possible for the brain tumor to have caused secondary changes in the pituitary with consequent manifestations of acromegaly.

The Relation of Acromegalia and Exophthalmic Goiter.—In most cases of acromegalia in the literature the thyroid gland was found more or less altered. It showed either hypertrophy or atrophy. In experimental extirpation of the pituitary body the thyroid seems to show transient hypertrophy followed by colloid degeneration. This condition can not be explained by the vicarious function of similar organs of the body. Cushing observed in most cases of acromegalia enlargement of the thyroid instead of atrophy, and he suggests the "same underlying biochemical factor" causing hyperplasia of both structures at the same time.

If the biochemical factor of Cushing's hypothesis is accepted, the hyperplastic condition of endocrine organs in our case is readily explained. But what is the "biochemical factor"? If the existence of this so-called biochemical factor is primary and the changes in the pituitary and other endocrine organs are secondary, acromegalia is not an independent disease but a partial manifestation of some pathologic metabolism that gives rise to a biochemical stimulus. If this exploitation of the relationship of the endocrine organs in our present case were accepted the mechanical pressure of the cerebral tumor would no longer be responsible for the production of the acromegalia. We are rather of the view that the hypertrophy of thyroid and other endocrine organs is secondary, due to functional alteration of the pituitary body. Although the thyroid gland and the pituitary body show histologic and functional similarity, there is no definite evidence that these organs necessarily function in a synergic manner. Both may show hypertrophy without a common biochemical stimulus, but in consequence of the altered function of one of them. It is possible that pathologically increased function of the anterior lobe of the pituitary gland exercises a certain influence on the secretory organs of the body, causing changes in these organs in the same direction.

We maintain that the cerebral tumor was, in all probability, the cause of the acromegalia in this case. First, the pituitary body was attacked showing both the clinical and pathologic picture of typical acromegalia. Second, the endocrine organs were involved in consequence of the altered function of the hypophyseal gland. The exophthalmic goiter is to be considered as a clinical manifestation of the pathologic thyroid gland.

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MOTOR DISTURBANCES IN LETHARGIC ENCEPHALITIS *

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We have seen that the main stress of the toxemia of lethargic encephalitis falls on the nerve cells. At certain sites the toxic action is reinforced by the direct attack of the infectious agents (Loewy-Strauss bodies?) on the nerve cells. The nerve sites attacked by these organisms are determined in part by the particular mucous membrane in which the initial invasion takes place. From this area, the organisms are carried along the lymphatics to the central nervous system. As the habitually invaded area is the nasopharyngeal mucosa, the nerve sites usually attacked are in the head end of the nerve axis; but if the intestinal mucosa be the invaded area, the brunt of the attack falls on the caudal end of the nerve axis. The direction, extent and severity of the attack on the nervous system determine the nature and degree of the resulting functional disturbances. It has already been stated that the more extensive the nervous mechanism on which a function depends, the more inevitable is the implication of that function in the attack. The nervous mechanism that subserves the function of movement is coextensive with the nervous system. In lethargic encephalitis movement is invariably deranged.

Lethargic encephalitis may affect the function of movement in all predictable ways, to all conceivable degrees and at all possible points of the motor mechanism. In different epidemics, and even during the same epidemic in different localities, or at different periods, particular varieties of motor derangement tend to prevail. In spite of such episodal variations among the qualities of the motor defect of lethargic encephalitis are certain that are banal and certain that at least in their emphasis are distinctive and pathognomonic.

Of any two movements, that which depends on the more extensive nerve mechanism is—*ceteris paribus*—the more liable to derangement in this infection. Where separate paths convey habitually correlated impulses to an associated group of muscles, sudden interference with any one path may disorganize temporarily the whole function. The

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initial result of the lesion may, therefore, be wholly disproportionate to its cause; and this disproportion may be accentuated if, as in the eye, the impairment of movement deranges another and more sensitive function.

MOTOR SYMPTOMS

Motor Disturbance of Eye.—The widespread nerve supply of the motor mechanism of the eye seldom wholly escapes implication in lethargic encephalitis. Double vision, near vision, blurred vision and other perceptual consequences may signify such motor implication even before it is detectable by skilled medical observation. The alternative paths of nerve impulses to the muscles of the eye may permit the speedy compensation of this functional disturbance. Hence, patients frequently neglect to mention it among their symptoms, unless specifically questioned regarding it.

In addition to these transient symptoms, lasting signs appear of impairment of the motor mechanism of the eyes. As a rule, the eyelids droop, first one, then the other. The droop is not commonly equal on both sides, and seldom completely closes both eyes; usually the palpebral fissures persist, one narrower than the other. In most cases the eyelids can be freely raised at will; but they fall again. Sometimes, however, they are not raised; but the patient then seems rarely to exert himself to open his eyes—the forehead is not wrinkled by his efforts.

The eyes assume the “rest” position; their axes being directed straight forward. They can be rotated as a rule into any normal position; but they do not maintain it; they tend to swing back to the middle line. Internal convergence is usually possible for a short time, at the end of which one eye rotates out again. The eyes may move deliberately and by stages, moving and halting and moving again, then swinging back to the position from which they started. Sometimes this movement is definitely cog-wheel, like the eye movement seen in Parkinson’s disease. Infrequently, definite but seldom conspicuous squints occur.

The pupillary reaction to light and accommodation may be lost or diminished. Usually both are unaffected. The pupils under a constant intensity of illumination contract and dilate, unable to maintain the pupillary posture corresponding to the illumination. A similar instability may be observable in accommodation.

Facial Muscles.—The facial muscles are often affected. Spasms, tics and fibrillation may sometimes be seen in them and occasionally nuclear paralysis. As a rule the facial affection consists chiefly of a loss of tone, a flattening of the muscles, with a fading of the wrinkles, and a lessening of the habitual facial folds. This tone

loss begins on one side and then spreads to the other. It is usually not complete or symmetrical; on one side it may affect the upper third, on the other the lower two thirds. It tends to disappear in emotional expression. It suggests a supranuclear palsy, but the apparent weakness may vanish in whole or in part on voluntary movement. Occasionally, even in patients who are not extremely apathetic, a partial or complete absence of emotional expression is observable in a face that otherwise shows no detectable motor defect. The facial weakness, no matter what its type may be, is seldom sufficient to lead to lacrimation, to drooling or to interference even with mastication.

Fifth Nerve.—Occasionally, the motor root of the fifth nerve suffers. During the irritative stage, chewing or grinding movements occur, and later lock-jaw may supervene. Even without premonitory movements, the jaw muscles may gradually stiffen till the mouth scarcely can be forced open. This irritative stage may be followed by a typical paralytic condition.

Tongue.—The tongue is implicated almost as frequently as the face. After a period of fibrillary twitching, the tongue on protrusion deviates to the weakened side; but this deviation bears no regular correspondence to any coexisting facial palsy, except in banal cases of hemiplegia. Later, the tongue is protruded with increasing difficulty or lies an inert or tremulous mass, immovable on the floor of the mouth. In such instances mastication and speech suffer.

The Soft Palate and the Constrictors of the Pharynx.—These may be so weakened as to render swallowing difficult or even impossible and to necessitate feeding by the stomach tube.

Cranial Motor Nerves.—Of the cranial motor nerves the eleventh (spinal accessory) is least commonly affected. When it is attacked, there may be wry neck but more frequently both sides are equally involved. Associated with the spasms, fibrillation, or loss of tone of the trapezeii and sternomastoids are similar affections of the other muscles of the neck region. Cephaloptosis or cephaloplegia, similar to that seen in poliomyelitis, is occasionally observed in lethargic encephalitis, especially in children.

As the cranial motor nerves are affected, in like manner the disease attacks the spinal motor nerves; and the consequences observed in the muscles of the head and neck are mutatis mutandi, observed in the muscles of the limbs and trunk. In the affected muscles spasms, fibrillations, weakness and loss of tone usually occur. The weakness may follow the spasms but oftener develops independently. Foot drop, wrist drop, sagging shoulder and other abnormal attitudes are assumed in the "resting" position. The dropped foot, wrist or shoulder, as a rule, can be raised at will; but it quickly drops again.

Hemiplegias and Monoplegias.—Not rarely banal hemiplegias or monoplegias occur. In these the distal muscles of the extremities are more involved than the proximal. Some power of movement usually persists in the trunk muscles of the affected side and in the muscles nearest the trunk in the affected limbs. The affected muscles are rigid and immobile. They assume the characteristic hemiplegic attitude, which is determined mainly by the dominance of the flexors over the extensors. Occasionally athetoid movements are seen in the paralyzed limbs.

Besides this banal hemiplegia, rigidity and loss of power of lesser degree occur, often hemiplegic in distribution. The rigid muscles, partially immobilized, tend to assume the typical hemiplegic posture and tremors appear in them. The characteristic tremor in lethargic encephalitis, like the tremor of Parkinson's disease, visibly affects extremities more than the head and trunk and is exaggerated by emotional excitement and by indirect intention. The lethargic encephalitis tremor, however, is not present "at rest"; it is markedly increased by direct intention and grows in amplitude as the objective of the movement is approached; it involves the forearm and wrist more than the hand and finger joints and the leg and ankle more than the toes; it is coarser, less symmetrical and more irregular than the typical Parkinsonian tremor. Where such a tremor ends, and a rhythmic movement purposeful in form but not in intent, begins, is difficult to define. The coarse pseudoparkinsonian tremor of lethargic encephalitis is scarcely distinguishable from a slight, irregular rhythmic purposeful movement.

In addition to this coarse tremor, the small, rapid tremor seen in most acute infectious diseases, occurs also in lethargic encephalitis.

Occasionally epilepsy develops.

The most characteristic of the motor disturbances is instability of posture.

LOSS OF MUSCLE TONE AND ITS RELATION TO POSTURE

Posture is the expression not of one, but of all the influences that determine muscle tone. The fundamental influence is the nutrition of the individual muscle cell, which is dependent partly on general nutritive conditions and partly on local and general control of these conditions through reflex and chemical action. In lethargic encephalitis loss of muscle tone occurs.

Part of this loss is analogous to that which occurs in all acute infectious diseases. Fever, toxemia, and their concomitant endocrine disturbances always reduce muscle tone. With the development of acute infectious fevers reflexes, such as the knee jerks, which depend on muscle tone, diminish or disappear; with the abatement of the

infection the muscle tone returns and the reflexes related to it recover their activity. Among infectious fevers lethargic encephalitis is conspicuous by its power directly to lower muscle tone, perhaps because of the virulence of its toxins, perhaps because of their inhibitory action on the pituitary and suprarenal glands.

Part of the loss may be due to inflammatory implication of nerve centers and paths by which the tone of the musculature is regulated. In lethargic encephalitis lesions may occur in the vestibular and cerebellar systems through which local, sectional and general changes in muscle tone are initiated and correlated. But the tone loss is common and such lesions are infrequent.

Part of this loss may be due also to the injury or destruction of the lower motor neuron, either at its origin in the nuclei of the anterior horn of the spinal cord and in their pontile and medullary analogues, or during its course through the peripheral motor nerve. A preliminary stage of irritation associated with muscular rigidity and fibrillation and myoclonia sometimes may be observed; then atonicity, atrophy and quantitative or qualitative changes in the electrical reactions occur. This loss is seldom widespread. It does occur, but it is often absent. Habitually, the electrical responses to faradism and to galvanism are unaltered. These responses are given by the fibrillar portion of voluntary muscle; which is therefore not blamable for the customary loss of muscle tone in lethargic encephalitis.

The tone loss is evident in the afflicted muscles even in so-called "rest" postures. There is in life no such thing as a posture of absolute "rest." "Rest" is a purely relative term. It signifies usually a posture of mental repose, the posture that is determined chiefly by the action of gravity. Its maintenance requires definite degrees of muscle tone. Hence, the afflicted facial muscles "at rest" flatten, the wrinkles fade and the usual facial folds disappear. In other words, owing to depreciation of muscle tone, the normal facial posture "at rest" is lost.

When a muscle contracts its tone increases. The increase of tone that accompanies voluntary movement may be adequate to mask the tone loss of the "resting" muscle. Thus, the atonic facial muscle sometimes moves at will with seeming perfection; but sometimes the flattening persists. The amount of disturbance persisting in movement measures the failure of the increased tonicity of the voluntary moving muscles to mask the tone loss of the "resting" muscle.

In moving, a muscle proceeds from an existing attitude through a series of intermediate postures to the desired attitude. To every posture pertains a definite muscular tone. Throughout a movement the tone of each posture must be maintained until that of its successor in the movement can be superimposed. Otherwise the desired

attitude is not directly attained and the movement is oscillatory or jerky. Lack of tone inherent to the intermediate postures leads to the phenomena seen when the eyes move—the slow rotations by stages and the persistent tendency to swing back to the “rest” position. It is as if the increased tone excited by the voluntary movement sufficed to attain the serial posture but not to preserve it. Hence, the eye swings to and fro in its rotation toward the desired goal.

And when the desired posture is attained by the eyes, lack of the necessary tone cuts short its duration. The eyelids can be raised at will but they fall again; and as often as they fall they can be raised once more only to fall once more. On internal convergence, an eye soon swings away. It may be brought in again only to swing out once more. With a constant source of light the pupil dilates and contracts. The outstretched arms may fall and be raised at will only to fall again.

This inability to maintain posture is not a fatigue manifestation, although fatigue may magnify it. It is not comparable with the similar phenomenon seen in myasthenia gravis. It is not a paralytic phenomenon. The movements can be indefinitely repeated and the same phenomenon is seen at the beginning as at the end of the test. The muscles show neither myasthenic nor atrophic electrical reactions. The lack of power to preserve posture is not due to the general lowering of tone for it affects only individual muscles or groups in particular areas.

In any movement certain muscles, agonists, contract, and they may be aided by the simultaneous contraction of others (synergists) which act similarly. Synchronously, the antagonistic muscles actively relax. If the antagonistic muscles do not adequately relax, the contracting agonists must first overcome the pull of the unrelaxed antagonists before movement can begin; and the movement proceeds by jerks and oscillations according to the momentary dominances of the agonists or antagonists in their contest. Such a struggle for dominance may occur. The evidence of it is spasms of the antagonistic muscles. Even without appreciable spasm there, the inability to maintain posture may exist. The force of gravity may suffice to destroy the posture attained.

When a posture is achieved it is maintained against gravity mainly by virtue of the tone inherent in the sarcoplasmatic portion of voluntary muscle. The sarcoplasmatic portion of voluntary muscle is distinguished from the fibrillar by its histologic structure, chemical composition, electrical reaction, function and nerve supply. The nerve supply is derived from neurons which originate in cells in the lateral horns of the spinal cord and their analogues in the cerebral end of the nerve axis. Through these nerves the sarcoplasmatic tone is

maintained on which depends the posture of the muscles. In lethargic encephalitis the nerve mechanism which subserves the sarcoplasmatic portion may be implicated, giving rise to their particular form of loss of power to maintain posture. Depression of the tone of the sarcoplasmatic portion of the agonists is probably the basis of this postural fault; but the tone of the antagonistic muscles may also be unduly exalted, for although spasm may be absent, the movements may nevertheless be somewhat stiff. At present, we do not know with certainty the precise seat of the defect. Investigation of it may afford valuable data regarding the nature of the relaxation of the antagonist and its relation to sarcoplasmatic tone.

It is noteworthy that the patient sometimes makes no effort to restore the lost posture. As there is no detectable sensory loss the lack of effort is probably due to mental apathy. This postural defect may arise when the lower motor neuron is demonstrably intact. It affects postures voluntarily assumed. There must exist, therefore, not one final common path to voluntary muscle, as Sherrington teaches, but two paths, namely, the lower motor neuron, its cell in the anterior horn and its white medullated fiber passing to the voluntary muscle, and the postural nerve of voluntary muscle. In poliomyelitis the lower motor neuron is mainly and commonly affected, and the postural nerve as a rule escapes. In lethargic encephalitis the lower motor neuron as a rule escapes and the postural nerve is mainly and commonly affected.

THE OCULOCARDIAC REFLEX (DAGNINI-ASCHNER PHENOMENON)—ITS USE IN MEDICINE AND PSYCHOLOGY

AN EXPERIMENTAL AND COMPARATIVE STUDY OF GROUPS OF NORMAL AND PATHOLOGIC SUBJECTS

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This phenomenon was first reported by Dagnini¹ at the meeting of the Academy of Medical Sciences of Bologna, June 17, 1908. Four months later B. Aschner,² unaware of the work of Dagnini, published a paper on the same subject. The reflex is called Aschner's reflex ordinarily, but in a spirit of fairness and justice to the Italian scientist, it should be identified primarily with the name of Dagnini.

The phenomenon consists in slowing of the radial pulse, lowering of the blood pressure and modification of the respiratory rhythm from compression of the eyeballs. Long before Dagnini and Aschner, Luciani had observed that stimulation of the branches of the trigeminal nerve with chloroform produced retardation of the heart; and Wagner von Jauregg used compression of the eyes to arouse stuporous patients. He did not explain the cause or the mechanism of the phenomenon.

Experiments on animals, carried out by Aschner, Miloslavich³ and others, showed that this was a real reflex having as the centripetal pathway the trigeminal nerve and as the centrifugal pathway the vagus. Aschner also demonstrated that the phenomenon could not be attributed to stimulation of the vagus by increased intracranial pressure.

Petzetakis⁴ and more recently Fumarola and Mingazzini⁵ have shown that the centrifugal pathway of the oculocardiac reflex is constituted also, although in a lesser degree, by the sympathetic. By

1. Dagnini, G.: *Intorno ad un riflesso provocato in alcuni emiplegici collo stimolo della cornea e colla pressione sul bulbo oculare*, Boll. di scienze med., Bologna **8**:380, 1908.

2. Aschner, B.: *Ueber einen bisher noch nicht beschriebenen Reflex vom Auge auf Kreislauf und Atmung. Verschwinden des Radialis pulces bei Druck auf das Auge (Vorläufige Mitteilung)*, Wien. klin. Wchnschr., No. 44:1529 (Oct. 29) 1908.

3. Miloslavich, E.: *Ueber Trigemini- Vagusreflexe*, Wien. med. Wchnschr. **51**:3051 (Dec. 17) 1910. Grossmann, J., and Miloslavich, E.: *Ueber die Beeinflussung der Hertztaetigkeit durch Bulbusdruck*, Wien. klin. Rundschau **12**:177, 1912.

4. Petzetakis: *Etude expérimentale sur les voies centrifuges du réflexe oculo-cardiaque*, Compt. rend. Soc. de biol. **76**:657 (April 25) 1914. *Effets de la section de la moelle cervicale sur le rythme cardiaque*, Arch. d. mal. du coeur **10**:66 (Feb.) 1917.

5. Fumarola, G., and Mingazzini, E.: *Contributo clinico e sperimentale allo studio del riflesso oculo-cardiaco*, Policlinico, Rome **24**:404 (Oct.) 1917.

severing the cervical cord, the sympathetic connection with the medulla is interrupted and the centrifugal impulse traveling only through the vagal cardio-inhibitory fibers causes exaggeration of the reflex.

Since the appearance of Dagainini's and Ascher's works a great number of papers have been published on this subject. Many reports have been contradictory and misleading and the inversion or exaggeration of the oculocardiac reflex was given too broad a significance.

The reflex has also been employed therapeutically in paroxysmal tachycardia (Lian,⁶ Voisin and Benhamou⁷) and in hiccough (Loeper and Weil⁸).

It has been assumed by some authors that the normal reflex is retardation of the pulse five to twelve beats per minute. When the pulse is reduced more than twelve the reflex is called exaggerated. When the retardation does not surpass four, the reflex is abolished. When instead of retardation, acceleration occurs, the reflex is inverted. Others have proposed similar classifications but, as will be shown later, classifications of this sort are arbitrary and do not give the real individual value because the same subject may show at different times a normal, an inverted and an abolished reflex, or a normal, an abolished and an exaggerated reflex. Small wonder that the reports of different authors are so discordant. Only in regard to tabes do the reports agree, the reflex having quite generally been found abolished by Auer,⁹ Fumarola and Mingazzini,⁵ Gautrelet,¹⁰ Lesieur, Vernet and Petzetakis,¹¹ Levine,¹² Loeper and Mougeot,¹³ Orlandi,¹⁴ Santiago Barabine¹⁵ and by the writer.

6. Lian, C.: De l'emploi thérapeutique du réflexe oculo-cardiaque dans les crises tachycardiques, *Arch. d. mal. du coeur.* **8**:193 (July) 1915.

7. Voisin, R., and Benhamou: La valeur thérapeutique du réflexe oculo-cardiaque, *Paris méd.* **9**:210 (March 8) 1919.

8. Loeper and Weil: Action favorable de la compression oculaire sur certaines manifestations nerveuses et en particulier sur le hoquet, *Bull. et mém. Soc. méd. d. hôp. de Paris* **37**:631, 1914.

9. Auer, E. M.: The Oculocardiac Reflex in Syphilis of the Central Nervous System, *J. A. M. A.* **68**:901 (March 24) 1917.

10. Gautrelet, J.: Le réflexe oculo-cardiaque, *Paris méd.* **3**:583, 1912.

11. Lesieur, Vernet and Petzetakis: Note sur l'abolition fréquente du réflexe oculo-cardiaque dans le tabes, *Bull. et mém. Soc. méd. d. hôp. de Lyon* **13**:198 (March) 1914.

12. Levine, S. A.: The Oculocardiac Reflex. An Electrocardiographic Study with Special Reference to the Differences Between Right and Left Vagal and Ocular Pressure in Tabetics and Non-Tabetics, *Arch. Int. Med.* **15**:758 (May 15) 1915.

13. Loeper, M., and Mougeot, A.: Absence fréquente du réflexe oculo-cardiaque dans le tabes, *Bull. et mém. Soc. méd. d. hôp. de Par.*, Dec. 26, 1913, p. 942; *Progrès méd.*, Dec. 27, 1913, p. 675.

14. Orlandi, N.: Sul valore clinico del riflesso oculo-cardiaco, *Riforma med.* **31**:232, 260 and 288, 1915.

15. Santiago-Barabino, A.: Contribucion al estudio del reflejo óculo-cardiaco, *Prensa med.*, Argentina, Buenos Aires, Nos. 29, 30, 31, 1917.

To give a few examples of the conflicting results in general paresis, Lesieur, Vernet and Petzetakis usually found the oculocardiac reflex exaggerated, while Fumarola and Mingazzini, Roubinovitch and Régauld de la Sourdière,¹⁶ and Aguglia¹⁷ found it abolished. In epilepsy Lesieur, Vernet and Petzetakis,¹⁸ Aguglia, and Dufour and Legras¹⁹ reported exaggeration of the reflex, while Maillard and Cordet²⁰ did not find it exaggerated and rejected the explanation of Lesieur, Vernet and Petzetakis that bromids cause the reflex to be less accentuated. In my experiments, although there was found a definite relative tendency to vagotonic reaction among the group of fifty epileptic patients, the findings of Lesieur, Vernet and Petzetakis could not be substantiated. Fumarola and Mingazzini⁵ found in epileptics a tendency toward exaggeration. Orzechowski and Meisels²¹ studied a group of epileptic patients with pharmacodynamic tests and found that they gave a vagotonic reaction. In diphtheria Aviragnet, Dorlencourt and Bouttier²² found that of twenty-six patients the reflex was normal in 42.3 per cent. and abolished in 57.7 per cent., while Gunson,²³ who examined fifty cases, found that the reflex was normal in 92 per cent. of the patients. In dementia praecox, Truelle and Bourdelique²⁴ found the reflex inverted or almost so. Roubinovitch

16. Roubinovitch and Sourdière, Regnauld de la: Le réflexe oculo-cardiaque dans les demences organiques, Soc. psych. de Par., June 18, 1914.

17. Aguglia, E.: Il riflesso oculo-cardiaco negli alienati di mente, Riv. ital. di neuropatol., psichiat. ed elettrot. **7**:385, 1914. Il riflesso oculo-respiratorio negli alienati di mente, Ibid. **8**:57, 1915.

18. Lesieur, Vernet and Petzetakis: Contribution à l'étude du réflexe oculo-cardiaque; son exagération dans l'épilepsie; ses variations sous l'influence d'actions médicamenteuses ou toxiques, Bull. et mém. Soc. méd. d. hôp. de Par. March 6, 1914; Considérations sur les modifications des reflexes produits par compression des globes oculaires chez certains epileptiques, Lyon méd. March 29, 1914, p. 721; Considérations physio-pathologiques sur un cas d'arrêt du coeur par le réflexe oculo-cardiaque chez un épileptique, Bull. et mém. Soc. méd. d. hôp. de Par. **37**:394 (March 6) 1914.

19. Dufour, H., and Legras, M.: Réflexe oculo-cardiaque provoquant l'arrêt du coeur, l'automatisme ventriculaire et la dissociation auriculoventriculaire; syndrome hypovarien et hyperthyroïdisme; crises épileptiformes; Bull. et mém. Soc. méd. d. hôp. de Par. **37**:686 (April) 1914.

20. Maillard and Codet: Le réflexe oculo-cardiaque chez les epileptiques, Soc. de Psychiat. de Par., June 18, 1914.

21. Orzechowski, K., and Meisels, E.: Untersuchung über das Verhalten des vegetativen Nervensystems in der Epilepsie, Epilepsia, Leipzig **4**:181 and 293, 1912-1913-1914.

22. Aviragnet, E. C.; Dorlencourt, H., and Bouttier, H.: Le réflexe oculo-cardiaque au cours de l'intoxication diphtérique, Compt. rend. Soc. de Biol., **76**:771 (May) 1914.

23. Gunson, E. B.: The Oculo-Cardiac Reflex, Brit. J. Child. Dis. **12**:97, 1915.

24. Truelle and Bourdelique: Le réflexe oculo-cardiaque dans la démence précoce des jeunes gens, Ann. méd. psychol. **7**:389, 1916.

and Regnaud de la Sourdière found the reflex abolished in 50 per cent. of their cases; in Graziani's report, 34 per cent. showed an abolished reflex, 48 per cent. a normal and 18 per cent. an inverted reflex. On the other hand, if we take the analysis of Gorriti,²⁶ based on a much larger number (207 cases), the results are entirely different: 84 per cent. had a normal, 8 per cent. an inverted and 8 per cent. an abolished reflex. Evidently in any variability curve, gaps are filled when a larger number of cases is studied. The report of Aguglia¹⁷ on seventy-two insane cases, gave 60.8 per cent. as showing an exaggerated reflex, while the report of Gorriti, based on 721 mental cases, showed that the oculocardiac reflex was normal in 81 per cent., inverted in 10 per cent. and abolished in 8 per cent. The reasons for such discordant results are to be found, aside from the individual differences, in the diversity of methods and scales employed.

ORIGINAL INVESTIGATIONS

I undertook a series of observations on different groups of subjects, normal and pathologic, for the purpose of comparing groups instead of individuals. The pathologic cases included tabes, general paresis, psychoneuroses, thyroid states, feeble-mindedness and a group of different organic nervous diseases. Most of these patients were taken from the Neurologic Institute, a few from the neurologic department of the Post-Graduate Medical School and Hospital, the feeble-minded from Randall's Island, through the courtesy of the medical director, Dr. Vavasour. Seventy-five of the 165 normal subjects were students in the psychologic department of Columbia University, the other ninety were taken at random regardless of age, sex and occupation.

The technic for the induction of the oculocardiac reflex is simple. The subject is placed in the recumbent posture or sits comfortably in an armchair, the head lying on the back of the chair. He is allowed to rest until the pulse becomes quite regular and equal for the four quarters of a minute. Then with the thumb and index or middle finger of the right hand gentle pressure is exerted on the eyeballs through the closed lids. The experimenter stands at the subject's right, taking the pulse at the right wrist. In order to obtain better results, especially when the oculocardiac has to be taken at different times, it is advisable to use an instrument similar to one which I shall describe later.

25. Graziani, A.: Contributo allo Studio del sistema endocrino simpatico in alcune psicosi, *Riv. ital. di neuropatol., psichiat. ed elettrotet.* **12**: Nos. 2 and 3 (Feb.-March) 1919.

26. Gorriti, F.: El reflejo oculo-cardíaco en 721 enfermos mentales, *Semana méd.* **23**:671 (Dec. 28) 1916.

In taking the oculocardiac reflex it is better to divide the pulsations into four periods of 15 seconds each. In this way one can easily stop whenever tenderness, or increased sensitiveness or a sharp fall of blood pressure appears and at the same time obtain the average for a minute. This method also gives a measure of the regularity of the oculocardiac reflex. Tabetic patients show no or slight variability in the four quarters of a minute whereas psychoneurotics show the greatest fluctuations; for instance, a psychoneurotic gives this response: pulse: 25, 26, 23, 24, total 98; oculocardiac reflex: 23, 26, 28, 27, total 104. A tabetic patient will give this response: pulse: 22, 22, 22, 22, total 88; or 22, 22, 22, 23, total 89; oculocardiac reflex, 22, 22, 22, 22, total 88; or 23, 22, 22, 22; total 89.

The existence of such great variations in the same person at different times shows the fallacy of designating the reflex by such adjectives as normal, normal feeble, normal medium, normal strong, exaggerated, inverted, when this designation is intended for diagnostic purposes. Moreover, the term "inverted" includes in the same category persons having an oculocardiac reflex from -1 to -20 or more; and the term "exaggerated" includes an oculocardiac reflex from $+13$ upward. Undoubtedly, the significance—physiologic, pathologic or psychologic—of an oculocardiac reflex of -1 and -20 or $+13$ and $+60$ must be quite different. Therefore, I suggest that the difference in one minute between the pulse rate without ocular pressure and the pulse rate with pressure be always indicated in full with a positive or a negative sign. This algebraic difference should be called reflex index (R. I.). The pulse rate should also be given, as the value of a reflex index is not absolutely the same in a bradycardiac as in a tachycardiac.

Two of the women on whom the experiment was performed—one a neurotic, the other a normal subject—fainted during the experiment; in three other cases the operation had to be stopped because of threatening arrest of the heart. In four epileptic patients pressure of the ocular bulbs caused a seizure.

Five hundred and one subjects were observed, 165 normal and 336 pathologic.

REFLEX INDEX IN NORMAL PERSONS

In Table 1 of my report three groups of normal subjects are reported, a total of 100 persons.

Section A contains fifty university students (male). Of these:

- 2 or 4 per cent. had a reflex index of 0
- 14 or 28 per cent. had a reflex index of 1, 2 (positive and negative)
- 13 or 26 per cent. had a reflex index of 3, 4, 5 (positive and negative)
- 12 or 24 per cent. had a reflex index of 6, 7, 8 (positive and negative)
- 5 or 10 per cent. had a reflex index of 9, 10, 11 (positive and negative)
- 4 or 8 per cent. had a reflex index of 12 or more (positive and negative)

Section B contains twenty-five students (female). Of these:

- 1 or 4 per cent. had a reflex index of 0
- 3 or 12 per cent. had a reflex index of 1, 2 (positive and negative)
- 9 or 36 per cent. had a reflex index of 3, 4, 5 (positive and negative)
- 8 or 32 per cent. had a reflex index of 6, 7, 8 (positive and negative)
- 2 or 8 per cent. had a reflex index of 9, 10, 11 (positive and negative)
- 2 or 8 per cent. had a reflex index of 12 and more (positive and negative)

Section C contains twenty-five male adults taken at random. Of these:

- 2 or 8 per cent. had a reflex index of 0
- 5 or 20 per cent. had a reflex index of 1, 2 (positive and negative)
- 7 or 28 per cent. had a reflex index of 3, 4, 5 (positive and negative)
- 6 or 24 per cent. had a reflex index of 6, 7, 8 (positive and negative)
- 1 or 4 per cent. had a reflex index of 9, 10, 11 (positive and negative)
- 4 or 16 per cent. had a reflex index of 12 or more (positive and negative)

If we were to classify the oculocardiac reflex as normal when the reflex index is from $+5$ to $+12$, as abolished when the index is from 0 to $+4$, as inverted when the index is negative, and as exaggerated when the index is above $+12$, the whole group of these 100 normal subjects would show:

- In 35 per cent. an abolished reflex
- In 33 per cent. a normal reflex
- In 9 per cent. an exaggerated reflex
- In 23 per cent. an inverted reflex

This group would look quite pathologic to those who expect to find an index of from $+5$ to $+12$ in normal subjects. Even more pathologic would appear the normal subjects of Table 2, if the same criteria were applied for classification of the oculocardiac reflex. In fact, we would have found the first time:

- 26 or 40.0 per cent. with an abolished reflex
- 13 or 20.0 per cent. with a normal reflex
- 9 or 13.8 per cent. with an exaggerated reflex
- 17 or 26.1 per cent. with an inverted reflex

And the second time:

- 36 or 55.6 per cent. with an abolished reflex
- 9 or 13.8 per cent. with a normal reflex
- 4 or 6.1 per cent. with an exaggerated reflex
- 16 or 24.6 per cent. with an inverted reflex

These figures give sufficient ground for rejecting any classification of the reflex index of the kinds that have been so widely accepted. Continuing in the analysis of Table 2, which includes sixty-five normal persons taken at random, whose oculocardiac reflexes were tested two or more times after an interval of from fifteen to ninety days, we found in the first test:

- 6 or 9.2% with a reflex index of 0
- 15 or 23.1% with a reflex index of 1, 2 (negative or positive)
- 17 or 26.1% with a reflex index of 3, 4, 5 (negative or positive)
- 13 or 20.0% with a reflex index of 6, 7, 8 (negative or positive)
- 3 or 4.6% with a reflex index of 9, 10, 11 (negative or positive)
- 11 or 16.9% with a reflex index of 12 and more (negative or positive)

In the second test:

- 11 or 16.9% with a reflex index of 0
- 22 or 33.8% with a reflex index of 1, 2 (positive and negative)
- 14 or 21.5% with a reflex index of 3, 4, 5 (positive and negative)
- 10 or 15.4% with a reflex index of 6, 7, 8 (positive and negative)
- 3 or 4.6% with a reflex index of 9, 10, 11 (positive and negative)
- 5 or 7.7% with a reflex index of 12 and more (positive and negative)

and in the third test, given to thirty persons, we find:

- 4 or 13.3% with a reflex index of 0
- 12 or 40.0% with a reflex index of 1, 2 (positive and negative)
- 9 or 30.0% with a reflex index of 3, 4, 5 (positive and negative)
- 3 or 10.0% with a reflex index of 6, 7, 8 (positive and negative)
- 0 or 0.0% with a reflex index of 9, 10, 11
- 2 or 6.6% with a reflex index of 12 and more (positive and negative)

Again in the first test we found:

- 6 or 9.2% with a reflex index of 0
- 42 or 64.6% with a positive reflex index
- 17 or 26.1% with a negative reflex index

and in the second test:

- 11 or 16.9% with a reflex index of 0
- 38 or 58.4% with a positive reflex index
- 16 or 24.6% with a negative reflex index

In regard to the variations found in the same person in Table 2:

- 9 or 13.8 per cent. kept the same sign
- 8 or 11.2 per cent. showed a difference from -1 to +1
- 3 or 4.6 per cent. showed a difference from -2 to +2
- 3 or 4.6 per cent. showed a difference from -3 to +3
- 6 or 9.2 per cent. showed a difference from -4 to +4
- 6 or 9.2 per cent. showed a difference from -5 to +5
- 2 or 3.1 per cent. showed a difference from -6 to +6
- 3 or 4.6 per cent. showed a difference from -7 to +7
- 25 or 38.5 per cent. showed a difference from ± 8 and more

The average index of the following subjects, who were tested from four to six times, gives an idea of the variability of a single index from the average.

- In Case 3, 6 tests yielded an average index of 4.83
- In Case 7, 4 tests yielded an average index of 2.5
- In Case 13, 4 tests yielded an average index of 5.5
- In Case 14, 4 tests yielded an average index of 11.25
- In Case 15, 6 tests yielded an average index of 1.1
- In Case 19, 4 tests yielded an average index of 2.25
- In Case 26, 4 tests yielded an average index of -1.0
- In Case 28, 4 tests yielded an average index of -0.5
- In Case 29, 4 tests yielded an average index of 1.75
- In Case 30, 4 tests yielded an average index of 4.5
- In Case 44, 5 tests yielded an average index of 1.2
- In Case 45, 6 tests yielded an average index of 1.0
- In Case 50, 4 tests yielded an average index of -4.5
- In Case 56, 4 tests yielded an average index of 4.5

This demonstrates how important it is to have the averages obtained from many trials.

In the absence of the ordinary statistic treatment of the tables reported, a measure of the number of times that each reflex index appeared amongst the normal subjects examined seems necessary before closing the report on Tables 1 and 2.

In the following tabulated summary the reflex indexes are given from 1 to more than 12 with their frequency as encountered in Tables 1 and 2 (first and second trials separately) and finally the frequency in all 230 tests.

It appears from this that the positive indexes are encountered about three times more frequently than the negative ones, and that the small indexes, 1 to 4 and 0, are the most frequent. The positive indexes from 1 to 4 and 0 indexes in the 230 tests constitute 41.3 per cent. of the whole group. This fact demonstrates once more that the so-called abolished reflex (reflex index from 0 to +4), is more frequently encountered in normal persons and therefore it cannot be considered pathologic.

REFLEX INDEXES FROM 1 TO MORE THAN 12, WITH THEIR FREQUENCY AS ENCOUNTERED IN TABLES 1 AND 2 AND THE FREQUENCY OF THE TWO HUNDRED AND THIRTY TESTS

R. I.	Table 1 5		Table 2 (First Test) 6		Table 2 (Second Test) 11		Total of 230 Tests 22	
	Positive	Negative	Positive	Negative	Positive	Negative	Positive	Negative
1	6	4	7	3	6	5	19	12
2	8	4	4	2	9	2	20	8
3	4	1	4	1	6	2	14	4
4	12	1	5	5	3	1	20	7
5	9	2	2	0	2	0	13	2
6	6	3	6	1	4	4	16	8
7	3	1	2	2	1	1	6	4
8	9	4	0	2	0	0	9	6
9	1	0	0	0	0	0	1	0
10	5	1	1	1	3	0	9	2
11	1	0	1	0	0	0	2	0
12	0	2	1	0	0	0	1	2
13 and more	8	0	10	0	4	1	22	1
Total	72	23	42	17	38	16	152	56

REFLEX INDEX IN PSYCHONEUROTIC AND FEEBLEMINDED PERSONS

A contrast of the findings of the normal subjects of Tables 1 and 2 with those Tables 3 and 4, which include, respectively, psychoneurotic and feeble-minded persons, does not reveal much.

In Table 3:

- 7 or 9.3 per cent. had a reflex index of 0
- 17 or 22.6 per cent. had a reflex index of 1, 2, positive or negative
- 16 or 21.3 per cent. had a reflex index of 3, 4, 5, positive or negative
- 17 or 22.6 per cent. had a reflex index of 6, 7, 8, positive or negative
- 7 or 9.3 per cent. had a reflex index of 9, 10, 11, positive or negative
- 11 or 14.6 per cent. had a reflex index of 12 and more

In Table 4:

- 3 or 6 per cent. had a reflex index of 0
- 12 or 24 per cent. had a reflex index of 1, 2, positive or negative
- 15 or 30 per cent. had a reflex index of 3, 4, 5, positive or negative
- 7 or 14 per cent. had a reflex index of 6, 7, 8, positive or negative
- 8 or 16 per cent. had a reflex index of 9, 10, 11, positive or negative
- 5 or 10 per cent. had a reflex index of 12 and more

If we classify the subjects of these two groups by the criteria used for the normal subjects, we do not find much difference, or rather these two groups look more normal than groups of Tables 1 and 2.

In fact, in Table 3:

- In 27 or 36.0 per cent. the reflex is abolished
- In 25 or 33.3 per cent. the reflex is normal
- In 5 or 6.7 per cent. the reflex is exaggerated
- In 18 or 24.0 per cent. the reflex is inverted

and in Table 4:

- In 18 or 36 per cent. the reflex is abolished
- In 21 or 42 per cent. the reflex is normal
- In 4 or 8 per cent. the reflex is exaggerated
- In 7 or 14 per cent. the reflex is inverted

The data of Table 4 do not agree with the report of Aguglia,¹⁷ who found the oculocardiac reflex most exaggerated in phrenasthenics. A difference is found in comparing the normal groups (Tables 1 and 2) with Table 8, which includes patients taken at random from the clinic suffering from organic nervous diseases. There is a larger number of cases with a 0 index in Table 8.

In Table 8 we find:

- 11 or 22 per cent. with a reflex index of 0
- 7 or 14 per cent. with a reflex index of 1, 2, positive and negative
- 15 or 30 per cent. with a reflex index of 3, 4, 5, positive and negative
- 7 or 14 per cent. with a reflex index of 6, 7, 8, positive and negative
- 6 or 12 per cent. with a reflex index of 9, 10, 11, positive and negative
- 4 or 8 per cent. with a reflex index of 12 and more, positive and negative

However, it must be remembered that among the organic cases also the reflex index is inconstant.

If these cases are classified into abolished, normal, exaggerated and inverted, as has been done with other groups, their distribution will not differ, to any sensible extent, from that found in classifying the results in sixty-five normal persons taken at random.

Here we have:

- 26 or 52 per cent. with an abolished reflex
- 13 or 26 per cent. with a normal reflex
- 4 or 8 per cent. with an exaggerated reflex
- 7 or 14 per cent. with an inverted reflex

REFLEX INDEX IN THYROID DISEASE

Omitting the cases of exophthalmic goiter in which the secretory alterations are qualitative rather than quantitative, the remainder of this group is constituted of thirty-four hyperthyroid and twenty hypothyroid cases.

Of the hyperthyroid cases

- 3 or 8.8 per cent. had a reflex index of 0
- 10 or 29.4 per cent. had a reflex index of 1, 2, positive and negative
- 4 or 11.7 per cent. had a reflex index of 3, 4, 5, positive and negative
- 8 or 23.5 per cent. had a reflex index of 6, 7, 8, positive and negative
- 0 or 0.0 per cent. had a reflex index of 9, 10, 11, positive and negative
- 9 or 26.5 per cent. had a reflex index of 12 or more, positive and negative
- 20 or 58.9 per cent. had a negative or 0 reflex index
- 14 or 41.1 per cent. had a positive reflex index

Of the hypothyroid cases

- 0 or 0 per cent. had a reflex index of 0
- 1 or 5 per cent. had a reflex index of 1, 2, positive or negative
- 4 or 20 per cent. had a reflex index of 3, 4, 5, positive or negative
- 2 or 10 per cent. had a reflex index of 6, 7, 8, positive or negative
- 4 or 20 per cent. had a reflex index of 9, 10, 11, positive or negative
- 9 or 45 per cent. had a reflex index of 12 or more
- 2 or 10 per cent. had a negative or 0 reflex index
- 18 or 90 per cent. had a positive reflex index

In this table and in Tables 6 and 7, in which more than one reflex index are given for one subject, the first was computed.

It was observed in many instances that the same subject showed a positive and a negative or 0 index at different times. Although a sharp distinction of the hyperthyroid and hypothyroid cases in sympathicotonic and vagotonic patient (Eppinger and Hess²⁷) cannot be drawn, it clearly appears from this table that the hyperthyroid patients tend to react as sympathicotonic and the hypothyroid patients as vagotonic. This fact may help somewhat in the diagnosis of the thyroid states. My results agree with those of Petzetakis,²⁸ Blanc,²⁹ and Garnier and Lévi-Franckel.³⁰ In several hypothyroid patients when large

27. Eppinger, H., and Hess, L.: *Pathologie des vegetativen Nervensystems*, Ztschr. f. klin. Med. **68**: 1919; *Die Vagotonomie, Eine klinische Studie*, Berlin, 1910.

28. Petzetakis: *Le reflexe oculo-cardiaque dans le syndrome hypothyroïdien*, Presse méd. **25**:12 (Jan. 8) 1917.

29. Blanc, J.: *La dysthyroïdie facteur de névrose. Le réflexe oculo-cardiaque régulateur de l'opothérapie thyroïdienne*, Progres méd. **32**:95 (March 24) 1917.

30. Garnier and Lévi-Franckel: *Modifications du réflexe oculocardiaque sous l'influence de la gestation*, Bull. et mém. Soc. méd. d. hôp. de Par. **37**:252 (July 24) 1914; *Le réflexe oculo-cardiaque dans la grossesse*, Compt. rend Soc. de biol. **76**:645 (April) 1914.

doses of thyroid extract were administered, the reflex index became smaller, sometimes even negative.

REFLEX INDEX IN EPILEPSY

On the patient in Case 47, suffering from jacksonian epilepsy, craniectomy was performed. Previous to the operation the reflex was $107-73 = +34$. Eleven months later the patient had a relapse, this time showing an epileptic equivalent. At this time the reflex index was $+16$.

Case 46 was also a case of jacksonian epilepsy; the patient was operated on. The two reflexes were taken five days apart, before and after the operation.

The assumption that bromids check the exaggeration of the oculocardiac reflex has not been proved in my cases. Subjects such as Case 11 showed a reflex index of $+19$ when taking bromids and $+9$ when not taking them; the patient in Case 2 had a reflex index $+9$ when taking bromids and $+3$ and -1 when not taking them; the reflex index of the patients in Cases 25, 27 and 50, who were taking bromids, were respectively $+13$, $+31$, $+13$, while those of the patients in Cases 3, 17, 18, 20, 22 and 28, who were not taking bromids, were 0, -5 ; -2 , -1 , -4 ; -2 ; -1 ; 0 and 0. The patient in Case 43 had a reflex index of -4 when taking bromids and -7 when not taking them. In Case 1 the reflex index was negative with and without bromids. Here also great variations were found in the reflex index when the oculocardiac reflex was taken at different dates. In general, it must be admitted that, despite the variations and the individual differences, the epileptic patients tend to react with a large positive index. The fact that not all epileptic patients are vagotonic demonstrates once more that epilepsy as a clinical entity does not exist; the convulsive seizure is the expression of entirely different pathologic conditions and etiologic factors.

In classifying the subjects of Table 6 we find:

- 4 or 8 per cent. with a reflex index of 0
- 10 or 20 per cent. with a reflex index of 1, 2, positive or negative
- 10 or 20 per cent. with a reflex index of 3, 4, 5, positive or negative
- 6 or 12 per cent. with a reflex index of 6, 7, 8, positive or negative
- 4 or 8 per cent. with a reflex index of 9, 10, 11, positive or negative
- 16 or 32 per cent. with a reflex of 12 or more, positive or negative

The average indexes obtained from the algebraic sum of the reflex index of this and of the other tables show that the epileptic and the hypothyroid patients have the largest average index, namely, $+7.48$ and $+11.95$, respectively. The smallest average index was found among the tabetic patients ($+1.23$).

REFLEX INDEX IN TABES AND GENERAL PARESIS

Thirty cases of tabes and twenty cases of general paresis are reported in Table 7. What differentiates these subjects, especially the tabetic patients, from all the others, is the small index, even when the oculocardiac reflex is repeated.

Of thirty tabetic patients, we find that in

- 8 or 26.6 per cent. the reflex index was 0
- 13 or 43.4 per cent. the reflex index was 1
- 5 or 16.6 per cent. the reflex index was 2
- 1 or 3.3 per cent. the reflex index was 3
- 3 or 10.0 per cent. the reflex index was 4 or more

And of twenty general paresis patients, we find that in

- 3 or 15 per cent. the reflex index was 0
- 6 or 30 per cent. the reflex index was 1
- 6 or 30 per cent. the reflex index was 2
- 1 or 5 per cent. the reflex index was 3
- 4 or 20 per cent. the reflex index was 4 or more
- 20 or 66.6 per cent. of the tabetic patients had a positive index
- 2 or 6.6 per cent. of the tabetic patients had a negative index
- 11 or 55.0 per cent. of the general paresis patients had a positive index
- 6 or 30.0 per cent. of the general paresis patients had a negative index

When the reflex was taken twice or more, the index did not constantly keep the same sign. Only in one case (general paresis) was the index greater than 12.

The absence of oculocardiac reflex in tabes and cerebrospinal syphilis has been attributed to bulbar lesions; to early involvement of the anastomotic fibers running from the fifth to the tenth cranial nerves in the medulla oblongata which have been described by Van Gehuchten and by Ramón y Cajal. If this were true, then we should find bulbar symptoms in all the cases of cerebrospinal syphilis showing a 0 or small index, because we could not imagine a lesion of the anastomotic fibers from the fifth to the tenth cranial nerves which would not sooner or later involve the surrounding fibers. Moreover, in cases of bulbar lesions the oculocardiac reflex has not been shown to be absent, as Guillain and Dubois³¹ have pointed out. These authors think that the oculocardiac reflex may serve to differentiate bulbar paralysis from the pseudobulbar. In several cases of bulbar paralysis that I have observed the oculocardiac reflex was not found absent all the time. Of course it will be absent when the center of the reflex arc is involved. It seems more probable that only in a small proportion of the cases of tabes, general paresis and cerebro-

31. Guillain, G., and Dubois, J.: L'abolition et l'inversion du réflexe oculocardiaque dans les paralysies pseudo-bulbaires, *Bull. et mém. Soc. méd. d. hôp. de Par.* **37**:584 (March) 1914.

spinal syphilis the absence of the oculocardiac reflex is due to a reflex lesion in the center; most cases of so-called absence of the oculocardiac reflex are due to peripheral lesions, when they are not due to constitutional factors.

Undoubtedly the syphilitic virus exerts a selective action on both subdivisions of the autonomic system, and when the receptors or the effectors are involved in the lesion, the reflex cannot take place or it will take place in an altered manner. The early digestive, sexual, secretory, and circulatory disorders, the changes in moods and in the affective and emotional life, which are observed in tabes, general paresis and cerebrospinal syphilis before the gross organic signs appear, are indications of the early involvement of the autonomic system. When the autonomic system is functionally affected, as in psychoneurotic patients, we find also cenesthetic, and similar functional signs suggestive of the unharmonious correlation between the two subdivisions of the autonomic system, such as myosis, enophthalmos and reduction of the palpebral fissures in some cases; mydriasis, exophthalmos and widening of the palpebral fissures in others. The conclusion which can be drawn from this consideration is that the oculocardiac reflex, when accurately studied in each person, may give useful information on the condition of the autonomic system and may thus help in the study of the personality.

It is obvious that from the clinical standpoint the oculocardiac reflex cannot constitute a positive diagnostic sign, which could compete with the other well defined and constant symptoms and laboratory findings of tabes. In the early stages of tabes, general paresis and cerebrospinal syphilis, the oculocardiac reflex could serve for differential diagnosis from functional nervous diseases, but the frequency with which the 0 or the small reflex index is encountered in normal persons renders this sign doubtful and unreliable.

FACTORS RESPONSIBLE FOR INDIVIDUAL DIFFERENCES AND VARIATIONS

The factors responsible for the individual differences and variations in the oculocardiac reflex are: (1) age, (2) sex, (3) position of the subject during the experiment, (4) amount of pressure exerted on the eyeballs, (5) physiologic state, (6) physical condition, (7) psychic conditions, and (8) internal or constitutional factors. By internal factors is meant mainly the makeup of the sympathetic endocrine system, which differs greatly among individuals. Broadly speaking, this system forms a large part of what is included in the old terminology of constitution, character and temperament. Besides its relations to other important vital functions, this system is at the basis of cenesthesia. The physical condition is indicated by the development, nutrition,

strength, etc., and in general by the state of health of the subject. The physiologic state includes such functions as digestion, fatigue, the action of drugs, etc., and menstruation or pregnancy in women. The other items do not require explanation.

Factors 1, 2 and 3 can be equalized easily.

Factor 4, which seemed to play a great rôle in the differences and variations found in the same person or in different persons, was eliminated with a simple device consisting of a small instrument resembling a lever having the fulcrum at the center and the power at the ends. The fulcrum was laid on the nose of the subject like spectacles whose lenses were substituted by two light wooden spoons protected with cotton, which could be moved toward the eyeballs with a gentle pressure. The pressure was applied with an ordinary sphygmomanometer having a long bag and a long band so that it could be wrapped around the head of the subject, covering the eyes. The reading in millimeters of mercury indicated the amount of pressure that was used for all subjects submitted to different tests. This method eliminated the errors that may occur when pressure is applied with the fingers. Roubinovitch³² has devised a similar apparatus, but he did not use the sphygmomanometer reading.

Factors 5 and 6 can be roughly equalized if a careful selection is made of subjects homogeneous as regards their physical and physiologic state.

Factors 7 and 8 cannot be equalized. Here the oculocardiac reflex may be of use. It may constitute an index of the psychic condition and of the sympathetic-endocrine makeup of the subjects, but only when the other factors have been standardized. In normal persons the reflex index may serve as an indication of reflex control, moods, courage, emotions, etc., traits whose scales are wanting. The different behavior of persons when confronted with the same situation is well known. Vague terms, such as temperament, character and neurotic constitution are used to explain why some persons are so abnormally self-conscious as to reach the degree of erythrophobia, why they cannot speak in public without showing a flushed face and having tachycardia, why they cannot undertake or meet a dangerous situation without trembling; whereas others never blush, meet crowds, without any

32. Roubinovitch, J., and de la Sourdière, Regnaud: Le réflexe oculocardiaque dans les hémiplegies et les diplegies cérébrales, *Bull et mém. Soc. méd. d. hôp. de Par.* **37**:909, 1914; Compresseur oculaire pour la recherche du réflexe oculocardiaque, *Comp. rend Acad. d. sc. Par.* **163**:137, 1916; *Progrès méd.* **35**:367 (Aug. 21) 1920. In the latter article the author describes a manometric oculocompressor which is a modification of the one he originated in 1916. When my paper was submitted for publication I did not know that such a modification was being meditated.

sign of anxiety and brave dangers without trembling. The answers to these questions have not yet been given, but it can be said without fear of contradiction that many doubts will be cleared up when a better knowledge of the autonomic system is obtained. For the study of this system, the Dagnini-Aschner reflex and probably also the Herring-Kratschmer reflex may give useful information. In order to obtain information of practical value, however, psychologic methods must be employed first on a large number of normal subjects before passing to the abnormal.

In Table 1, height and weight are given. These measurements, together with other minute anthropometric data not given, were taken in order to learn whether any correlation exists between the morphologic type of subject and the oculocardiac reflex. It seemed to the writer that subjects having small positive, or a 0 or a negative index were more intelligent than those who had a larger positive index. An intelligence test (Otis, form A and Alpha) was given to forty persons of group A (Table 1). Realizing the inconstancy of the reflex index, the results of these correlations cannot be taken at face value. When the average of the indexes, based on many tests, are obtained, the correlation between the average index and the intelligence, and between the average index and the morphologic type, will form the subject of another paper.

INFLUENCE OF CRANIAL NERVES ON REFLEX INDEX

Before concluding I shall report what seems to be an experimental and clinical confirmation of the experiments of Aschner on animals. In order to find what cranial nerves have an influence on the oculocardiac reflex, a group of patients presenting unilateral complete peripheral paralysis of cranial nerves was selected. The oculocardiac reflex was tested at different times on one case each of paralysis of the third, fourth, fifth (motor branch) cranial nerves; fifth (sensory branch) sixth, ninth, eleventh and twelfth cranial nerves; on three cases of second and on eight cases of seventh cranial nerve paralysis. No case of eighth and tenth cranial nerve paralysis was available. In a case of diphtheric polyneuritis, in which both vagus nerves were involved, the index was found to be very large. The patient, a woman 27 years of age, showed left facial paralysis, absolute pupillary rigidity, paresis of the muscles of respiration which kept the patient in severe danger of death for several days, bilateral paralysis of the velum palati, disturbance of deglutition, and laryngeal paralysis. The first time the oculocardiac reflex was $112-64=+48$; three days later it was $130-58=+72$. As there was danger of arrest of the heart and of respiratory paralysis, the reflex was not repeated again until four weeks later when the patient's recovery was assured. The reflex

index at that time was between $+6$ and $+12$. Collet and Petzetakis,³³ in five cases of traumatic vagus lesion, found the reflex inverted. In the case referred to in the foregoing it must be supposed that the thoracic or inferior cardiac branches of the vagus nerves were intact, while the cardiac sympathetic branches were affected, causing the reflex to discharge itself through the vagus alone with production of marked bradycardia.

In the cases of second, third, fourth (motor), fifth, sixth, seventh, ninth, eleventh and twelfth nerve paralysis, no substantial differences were found between the reflex of the left eye and the right eye. The difference was never above 2 counts negative or positive.

In three cases of Bernard-Horner syndrome, differences of a few counts between the right and left index were found. Probably the differences would have been larger if the lesion of the cervical sympathetic nerve had been complete.

The most important observation was made on a patient suffering from "tic douloureux," who was operated on by Dr. Taylor at the Neurologic Institute for posterior root resection of the right trigeminal nerve. After the operation the reflex of both eyes was taken four times, once every second day. The pulse rate is given for quarters of a minute. The findings of the first examination were: 27, 27, 27, 27, average 108; oculocardiac reflex, right, 27, 27, 27, 27, average 108; left, 26, 25, 26, 25, average 102. The findings of the second examination were: pulse 18, 17, 17, 17, average 69; oculocardiac reflex, right, 17, 17, 17, 17, average, 68; left, 15, 15, 15, 15, average 60. The findings of the third examination were: pulse 19, 20, 21, 21, average 81; oculocardiac reflex, right, 20, 19, 20, 21, average 80; oculocardiac reflex, right, 20, 19, 20, 21, average 80; left, 18, 18, 18, 18, average 72. The findings of the fourth examination were: pulse, 23, 21, 20, 20, average 84; oculocardiac reflex, right, 22, 21, 21, 20, average 84; left, 20, 18, 18, 19, average 75.

INFLUENCE OF PAIN STIMULATION ON REFLEX INDEX

Pain stimulation with heavy pressure on the mandible, maxilla and eyebrow gave these results in three trials: left mandible, 20, 21, 21, 21, average 83; left maxilla, 21, 21, 21, 22, average, 85; left eyebrow, 20, 20, 21, 21, average 82; right mandible, 20, 20, 21, 21, average 82; right maxilla, 20, 21, 22, 20, average 83; right eyebrow, 20, 21, 21, 21, average, 83.

This experiment was tried in order to exclude the possibility that

33. Collet and Petzetakis: Le réflexe oculo-cardiaque dans les lésions-traumatiques des pneumogastriques, *Compt. rend. Soc. de biol.* **79**:1147 (Dec. 16) 1916.

the slowing down of the pulse rate, obtained by compression of the right eye, was merely a pain response, which could not take place on the right on account of anesthesia. The same experiment was tried on thirty normal subjects (Table 2) and on thirty pathologic subjects, but no changes in the pulse rate similar to that obtained through ocular compression were found. This experiment proves that the oculocardiac reflex is not a response to pain stimulation; while the case of section of the fifth cranial nerve confirms the existence of a real reflex as described by Dagnini and Aschner. In this case the reflex could not be obtained on account of interruption of the centripetal pathway.

SUMMARY

1. The oculocardiac reflex is subject to individual differences and variations, as is the pulse.

2. Since normal persons are subject to the same changes in their oculocardiac reflex that have been found in many pathologic conditions, the oculocardiac reflex cannot constitute a positive sign for differential diagnosis. It may serve only as an indicator of probability.

3. The classification of the oculocardiac reflex into normal, abolished, inverted and exaggerated classes cannot be accepted on account of the extreme inconstancy of the reflex index: the same normal or abnormal subject may present a positive, a negative, and a 0 index at different times, even when the hour, position, and the amount of ocular compression are kept constant.

About 40 per cent. of the normal subjects examined by the writer showed a reflex index of from 0 to +4.

4. For clinical and psychologic purposes, the algebraic difference between the pulse rate without ocular compression and the pulse rate during the ocular compression should be given instead of such terms as normal, abolished, inverted and exaggerated. The algebraic difference should be termed the index of the oculocardiac reflex.

5. No scale of the oculocardiac reflex can be reliable which is not expressed in terms of averages. No value can be attributed to an index obtained at a single test. To measure a variable fact, such as the oculocardiac reflex, many measurements are necessary. Therefore the reflex index of a normal or pathologic person should be obtained only from the average of several tests taken at different times. Slight deviations of a few counts above or below the average index should not be given a pathologic significance. Large deviations from the reflex index, although likely to be found in normal states and in normal subjects, may indicate abnormal conditions of physiologic or psychologic states and may also be the expression of pathologic states.

6. In psychology the oculocardiac reflex may be of use for the study of some traits the scales of which are lacking, if a large number of cases and proper methods are used.

7. My researches on pathologic cases have shown that in tabes the reflex index is 0 or very small; exceptionally it surpasses three units; it shows slight variations or no variations at all when taken taken at different times.

In general paresis the index tends to remain small, but cases showing a larger positive or negative index are encountered with much more frequency than in tabes.

In the feeble-minded no tendency to a large positive index (exaggerated reflex) was found, as reported by some authors. The groups of psychoneurotic persons, feeble-minded persons and persons with organic nervous diseases did not show any substantial variations that could not be found in a group of normal subjects.

In epilepsy, although a well defined tendency toward a large positive index (vagotonic reaction) was found, it was not the rule; cases with small positive indexes, with 0 and negative indexes, were quite often encountered. Bromids did not reduce the reflex index. Large variations from positive to negative indexes were found in the same person at different times.

In thyroid states, a definite tendency was found on the part of the hypothyroid patients to react with a positive index and of the hyperthyroid patients to react with a negative index; this tendency was much more definite in hypothyroid patients. Where the reflex could be repeated at different intervals, large variations were found. Administration of thyroid extract to the hypothyroid patient seemed to produce a reduction of the index.

8. In normal and pathologic cases, pressure over different sensitive spots of the body did not induce the changes in the pulse rate that were obtained with ocular compression.

9. Unilateral paralysis of the second, third, fourth, fifth (motor branch), sixth, seventh, ninth, eleventh, and twelfth cranial nerves did not modify the reflex to a substantial extent. Involvement of the vagus nerves greatly influenced the reflex index; involvement of the cervical sympathetic nerve caused a slight alteration of the index. Resection of the sensory branch of the trigeminal nerve produced suppression of the reflex on the side of the lesion, without influencing the reflex index of the other side. These facts substantiate the results of experiments previously reported, namely, that the centripetal pathway of the oculocardiac reflex is constituted exclusively by the sensory branch of the trigeminal nerve and that the centrifugal pathway is constituted mainly by the vagus and partially by the sympathetic nerve.

PHENOLSULPHONEPHTHALEIN ABSORPTION FROM THE SUBARACHNOID SPACE IN PARESIS AND DEMENTIA PRAECOX

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The study of absorption of dye from the subarachnoid space is not new. The use of phenolsulphonephthalein is, however, of comparatively recent origin. Dandy and Blackfan¹ used it in their studies of hydrocephalus. (The solution used was neutral in reaction and nontoxic. The solution prepared for kidney function tests should not be used because of its alkaline reaction.) In thirty to fifty minutes after the dye had been injected into the subarachnoid space, traces of it were found in the right lymphatic and thoracic ducts whereas the dye appeared in the blood in three and in the urine in six minutes. In two hours from 35 to 60 per cent. of the dye was recovered in the urine. They conclude that the cerebrospinal fluid passes directly into the blood, and the lymph spaces are not concerned with its absorption. Shortly after the dye had been injected into the lumbar subarachnoid space, it was found in the cerebral ventricles.

Mehrtens and West² used phenolsulphonephthalein in a series of cases and reported their results in 1917. They injected 1 c.c. of the neutral dye into the lumbar subarachnoid space, washed the dye from the needle with 3 c.c. of previously withdrawn spinal fluid, thus preventing its escape into the muscles along the needle track when the needle was withdrawn; they catheterized the bladder and determined the time of appearance of the dye in the urine. Quantitative estimations of the amount excreted varied greatly, and the authors suggest that the dye may suffer considerable reduction before complete elimination can take place. Some of the spinal fluids showed a reduction of from 10 to 20 per cent., while others showed no reduction at all. They observed that diseases of the central nervous system, especially when involving the meninges, produce a lengthening of the appearance time to as much as seventy minutes in some cases. In syphilis a lengthening of the appearance time may be produced before any other evidence of central nervous system involvement has appeared, and they conclude that at present no definite conclusion can be drawn as to the exact location of the absorbing tissue.

1. Dandy, W. E., and Blackfan, K. D.: An Experimental and Clinical Study of Internal Hydrocephalus, *J. A. M. A.* **61**:2216 (Dec. 20) 1913.

2. Mehrten, H. G., and West, H. F.: *Arch. Int. Med.* **20**:575 (Oct.) 1917.

Incidental to the routine examination of the spinal fluid in a number of cases of paresis and catatonic dementia praecox, observations were made by the writer on the absorption of the phenolsulphonephthalein from the subarachnoid space.

TECHNIC

Patients were placed on their left side with the buttocks and shoulders on the same level. The skin was cocainized; lumbar punctures made between the third and fourth lumbar vertebrae and a stopcock connected with the needle. Three c.c. of fluid were collected under sterilized paraffin oil for the determination of specific gravity, hydrogen-ion concentration, colloidal gold and globulin tests and the Wassermann reaction. A rubber tube 5 cm. in length with adapters and connected with a 30 c.c. Luer syringe barrel was then attached to the stopcock, and 15 c.c. of spinal fluid were allowed to flow into the syringe barrel. The rubber tube was closed by kinking it on itself and then detaching it. A 2 c.c. tuberculin syringe containing 1 c.c. of a sterilized, neutral solution of phenolsulphonephthalein having a specific gravity of 1.0061 was connected with the stopcock and the dye slowly injected. The syringe was disconnected and the rubber tubing leading from the 30 c.c. syringe was attached to the stopcock. The 15 c.c. of previously withdrawn spinal fluid were slowly injected by controlling the pressure on the piston so that two minutes were required to empty the syringe. In this way the dye was thoroughly washed from the needle. Immediately after the needle was withdrawn the patient was placed on his back, the bladder catheterized and the time of appearance of the dye determined by collecting the urine in a test tube containing a few drops of 10 per cent. sodium hydrate solution.

Three or more days after the intraspinal injection, the bladder was catheterized and as soon as it was empty, 1 c.c. of the neutralized dye was injected deep into the deltoid muscle and the time of its appearance in the urine determined. The stopcock made it possible to withdraw fluid and inject the dye without losing a drop of either.

DISTRIBUTION OF THE DYE

It was necessary first to determine the average amount of cerebrospinal fluid in cases of dementia praecox and paresis. Such information was obtained from cadavers. Immediately after death, with the body on its back and the head slightly elevated, a needle was inserted into the cisterna magna and another into the lateral ventricle, a small hole having been bored through the skull for the purpose. The fluid was collected in a graduated cylinder and the head rotated

several times until no more fluid flowed from the cisterna. The body was then turned on its side with shoulders slightly elevated and a needle inserted between the twelfth thoracic and first lumbar vertebrae. The fluid flowing from this needle and representing the amount in the canal from the medulla to the end of the cord was collected and measured. When no more fluid flowed, a fourth needle was inserted between the last lumbar and first sacral vertebrae. Fluid flowing from this needle represented the amount in the lumbar area. The average amount of fluid in sixty dementia praecox cases of all forms was 110 c.c. in the skull, 15 c.c. in the thoracic part of the cord and 15 c.c. in the lumbar area. From twenty-eight cases of paresis the amounts were 135, 18 and 15 c.c., respectively.

TABLE 1.—MAXIMUM AND MINIMUM AMOUNTS OF FLUID FOUND

	Maximum Amount in C.c.			Minimum Amount in C.c.		
	Skull	Thoracic Part of Cord	Lumbar Area	Skull	Thoracic Part of Cord	Lumbar Area
Dementia praecox	120	18	18	90	13	12
Paresis.....	150	19	19	110	13	13

The specific gravity of the fluid from the cases observed (determined by pyknometer) varied from 1.0061 to 1.0063 in the dementia praecox group and from 1.0061 to 1.0073 in the parietic group. The specific gravity of the dye was 1.0061. One would therefore expect the dye to diffuse slowly in those cases in which the specific gravity of the fluid was the same as that of the dye solution and to rise to the higher portions of the cord when the dye solution was lighter than the spinal fluid.

A. E. Barker³ studied frozen sections of cadavers and found that with the body on its back, the highest point in the spinal canal was in the cervical region and next to this, the space between the third and fourth lumbar vertebrae. The canal slopes in both directions from this interspace. The cephalic slope continues downward to the fifth and sixth thoracic vertebrae when it begins to run upward again until the third cervical vertebra, or if the head is on a pillow, until the foramen magnum is reached. It would, therefore, seem that the most appropriate place to inject the dye would be between the third and fourth lumbar vertebrae, for this would give it an opportunity to flow in both directions and cause greater diffusion. One would expect to find the dye as far up as the fifth or sixth thoracic vertebrae in a few minutes. Dandy and Blackfan state that the dye finds its way into the cerebral ventricles a few minutes after it is injected into the lumbar subarachnoid space.

3. Barker, A. E.: *Brit. M. J.* 1:597, 1912.

In order to determine how far the dye diffused, 1 c.c. was injected as described above into the lumbar subarachnoid space of a cadaver immediately after death. Ten minutes later the cisterna magna was punctured, the buttocks elevated so that the spinal canal was at an angle of about 45 degrees, and 20 c.c. of clear fluid withdrawn before any dye appeared. In another instance puncture between the fifth and sixth thoracic vertebrae with the body placed horizontally yielded clear fluid—no dye was present. These experiments were repeated on other cadavers one hour after injection of the dye, with the same results. The conclusion is that the dye does not leave the lumbar area in cadavers. In a selected number of dementia praecox and parietic cases puncture of the cisterna magna, after injection of the dye into the lumbar subarachnoid space, yielded clear fluid free from traces of color. Punctures were made, one in each case, at intervals of one-fourth, one-half, one, three and five hours after the injection of the dye. In these cases there was no diffusion upward of the dye as far as the cisterna. It, therefore, remained in the lumbar or thoracic regions or was destroyed or absorbed before it could reach the cervical region.

To determine whether the cerebrospinal fluid destroyed the dye, its effects were studied in the test tube. Because of the above findings, it was assumed that all the unabsorbed dye was in the lumbar and lower thoracic regions, and, since there was an average of 20 c.c. of fluid in these areas (15 c.c. in the lumbar area plus one-third of the amount in the whole thoracic area), each cubic centimeter contained approximately one-twentieth c.c. of the dye. This amount of dye was then added to 1 c.c. of freshly drawn fluid collected under sterilized paraffin oil and the same amount was added to 1 c.c. of sodium carbonate solution having a p_H of 7.4, which was the same as that of the spinal fluid. The tubes were covered with sterilized paraffin oil, closed with cork stoppers and placed in the dark at 37 C. for twenty-four hours. At the end of this time one-twentieth c.c. of the dye was added to a freshly prepared sodium carbonate solution having a p_H of 7.4. Two c.c. of 5 per cent. sodium hydrate solution were then added to each tube and their contents compared in the Kober colorimeter. The freshly prepared sodium carbonate solution was used as the standard. There was no determinable destruction of the dye by the spinal fluid in any instance. Mehrtens and West² found a reduction of 10 to 20 per cent. in some cases and no reduction at all in others.

The dye was therefore not reduced by the fluid in a test tube under the above conditions and perhaps was not reduced by it in the spinal canal. Quantitative estimations of the amount of dye elimi-

nated were not made. The observations that have been made indicate that the dye is absorbed from the lumbar region and that it does not reach the cisterna in five hours.

APPEARANCE TIME

Dandy and Blackfan found the dye in the urine six minutes after it had been injected into the lumbar subarachnoid space. The tables in the paper of Mehrrens and West give the appearance time as four to ten minutes—in one case fourteen minutes. These figures, of course, are for normal subjects or at least for subjects not having demonstrable involvement of the central nervous system.

TABLE 2.—DEMENTIA PRAECOX CASES

Number	Age	Duration, Years	Appearance Time, Minutes	
			Intraspinal	Intramuscular
9985	30	6	25	15
5992	44	13	25	12
6068	34	15	28	13
8529	27	6	30	20
8278	29	7	30	11
8477	34	10	30	15
4000	51	25	30	10
5548	38	15	35	6
8672	27	7	37	18
5220	44	20	37	10
10131	27	3	37	12
7938	35	15	40	7
6209	37	19	41	10
6386	39	20	42	7
6900	30	11	45	4
8229	28	8	45	11
6043	33	5	48	6
7331	34	12	50	10
9659	26	3	53	9
9056	26	5	55	13
8916	28	5	58	10
5145	42	16	60	11
8622	24	6	65	17
5975	33	13	80	11
7516	30	10	90	7
5945	33	14	96	10
7903	30	10	100	8
10564	19	1	104	10

PARESIS CASES

10150	37	4	12	18
10719	34	1	13	9
10675	33	3	25	15
10716	46	2	25	10
9315	38	4	26	14
10189	34	2	28	18
10503	54	1	28	19
10525	39	2	30	14
10647	39	2	31	10
9550	39	5	32	12
10621	49	1	33	20
10689	39	1	34	12
8298	43	7	35	14
10724	47	2	40	10
10654	52	2	42	12
10085	31	3	51	17
10414	40	4	68	9

From the table it will be seen that the shortest appearance time in the series was twelve minutes. It occurred in a case of paresis. The longest appearance time was in a case of catatonia, 104 minutes. In the paretic cases the time varied from twelve to sixty-eight minutes and in the dementia praecox group from twenty-five to 104 minutes. After intramuscular injection, the appearance time varied from four to twenty minutes.

SUMMARY

One c.c. of neutral, sterilized solution of phenolsulphonephthalein having a specific gravity of 1.0061 was injected into the lumbar subarachnoid space and the contents of the needle washed into the canal with 15 c.c. of previously withdrawn spinal fluid. The time of appearance of the dye in the urine was then determined. Twenty-eight cases of catatonic dementia praecox and seventeen cases of paresis were observed. Age, duration of the psychosis, physical and mental condition, so far as could be determined, had no constant effect on the appearance time. In all cases the dye was longer in making its appearance in the urine than the normal time stated by Dandy and Blackfan, six minutes, and by Mehrrens and West, four to ten minutes. In these observations the time varied from twelve to sixty-eight minutes in the case of paresis and from twenty-five to 104 minutes in the case of catatonic dementia praecox.

The specific gravity of the spinal fluid varied from 1.0061 to 1.0063 in the dementia praecox group and from 1.0061 to 1.0073 in the paretic group. The hydrogen-ion concentration in all cases was 7.4. The Wassermann reaction, gold and globulin tests were all negative in the dementia praecox cases and were positive in the paretics.

The dye was not found in fluid drawn from the cisterna magna at any time up to five hours after it had been injected into the lumbar subarachnoid space. The absorption of the dye took place from the lumbar region.

THE FALSE LOCALIZING SIGNS OF SPINAL CORD TUMOR

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The development of the surgery of tumors of the spinal cord and membranes has been due to the advances in our knowledge of the fiber tracts and their pathways and of the localization of functions in the different tracts and segments of the cord. With these advances the names of Bechterew, Cajal, Koelliker, Lenhossek and others, and of Brown Séquard, Sherrington, Thorburn, Mackenzie, Allen Starr and Head are indelibly associated.

Although much is still unknown, it is now possible to diagnosticate the level of a spinal new growth, the side of the cord on which the tumor lies, and the relation of the growth to the cord and to the structures which surround it. Thus, in a large number of cases in our experience, it has been possible to determine before the operation whether the tumor was intramedullary or extramedullary; whether it lay in front or behind, to the left or to the right of the cord, and whether it lay behind or in front of one or more nerve roots.

The diagnosis "spinal cord tumor" can be correctly made in the majority of instances, if a careful history of the beginning and progress of the symptoms is obtained, and the examination is a thorough one. It is unusual to find a tumor when none is expected, and it is becoming increasingly rare wrongly to make the diagnosis "spinal cord tumor." We are accustomed to divide the patients into three groups: (1) those in whom the diagnosis can be made with certainty, (2) those in whom a tumor is probable, and (3) those in whom a tumor is possible but not probable. In the patients in the first group a tumor was usually found; in those of the second group, it was often present, and in those of the third group the disease was rarely a new growth.¹

Regarding the question of the correct level diagnosis, we have had, as reported elsewhere,² some patients in whom the signs were wrongly interpreted, or the examination was not made with sufficient care, so that the tumor was first looked for several segments higher or lower than where it was finally found. In other patients, without doubt, the level diagnosis was correct, but the desired segments of

1. In my eighty-one operations for spinal cord tumor, the correct diagnosis was made seventy-three times; in the other eight patients a tumor was considered one of the possibilities.

2. Elsberg, C. A.: Concerning Spinal Cord Tumors and Their Surgical Treatment, *Am. J. M. Sc.* **159**:194 (Feb.) 1920.

the cord were not at first exposed by the laminectomy, either because the wrong vertebral arches were removed or on account of the natural variations in the relation between the vertebrae and the cord segments. In not a few cases, several more laminae had to be removed before the new growth was exposed. With increasing experience these mistakes have become more rare, but I am conscious that others might have done better than I have done.

In some patients, however, the symptoms and signs noted at repeated examinations by different examiners pointed clearly to a certain level and a definite location, but the growth was found to have entirely different relations to the cord. I have characterized these as cases with "false localizing signs."

CHANGES IN THE SENSORY LEVEL SIGNS OF SPINAL TUMORS

The following histories are given as examples of this phase of our subject:

CASE 1.—History.—There were increasing spinal symptoms of sixteen months' duration and level symptoms at the seventh thoracic segment. Laminectomy was performed. No tumor was found. Four years later, level symptoms at the seventh and eighth cervical segment appeared. Laminectomy was performed, and an extramedullary tumor was removed.

Mrs. L. G. was admitted to the New York Neurological Institute as a private patient of Dr. Foster Kennedy in June, 1919. She was 35 years of age, married, and had three healthy children. During a period of fourteen years, she had sprained her right ankle a number of times. In February, 1910, she had an attack of influenza, after which she noticed that her left lower extremity felt heavy and that she frequently had a tingling sensation in the left leg and foot; soon after the right lower extremity became similarly affected. Two months after the onset of her symptoms, the patient began to have decided difficulty in walking; the limbs became progressively weaker during the fall and winter of that year. She wore a brace on the right ankle on the theory that the pain and weakness were due to the repeated sprained ankle. The right leg was always weaker than the left, but the tingling was most marked in the left leg. In the spring of 1911, she began to have pain in the back. She was treated for flat feet, and arch supports were ordered for her; these did not improve the condition. At about this time she first noticed a loss of feeling in her lower limbs. The limbs became more and more stiff and weak and she became bedridden; she had some difficulty with micturition.

First Examination.—June 16, 1911: The patient was well nourished. The cranial nerves were normal; the pupils reacted well to light and accommodation; nystagmus was not present. Upper Extremities: Power was good, the right equal to the left; biceps, triceps and wrist reflexes were active and equal. Sensation was normal in both extremities. Abdominal Reflexes: These could not be obtained (the patient was stout). Lower Extremities: Both lower extremities were spastic and weak, the right much weaker than the left. The patient had greater difficulty in raising the right limb from the bed than the left one. Flexor power at the knee and ankle was also much weaker on the

right side. Knee and ankle reflexes were much exaggerated and double ankle clonus and the Babinski reflex were present. The extension of the large toe was more marked on the right.

Sensory Status (Fig. 1): Sensation in the chest and upper limbs was normal. Posteriorly on the left side, below the level of the ninth thoracic spine there was almost complete loss of tactile, pain and temperature sensibility, while anteriorly the same condition obtained up to the same level. Over the

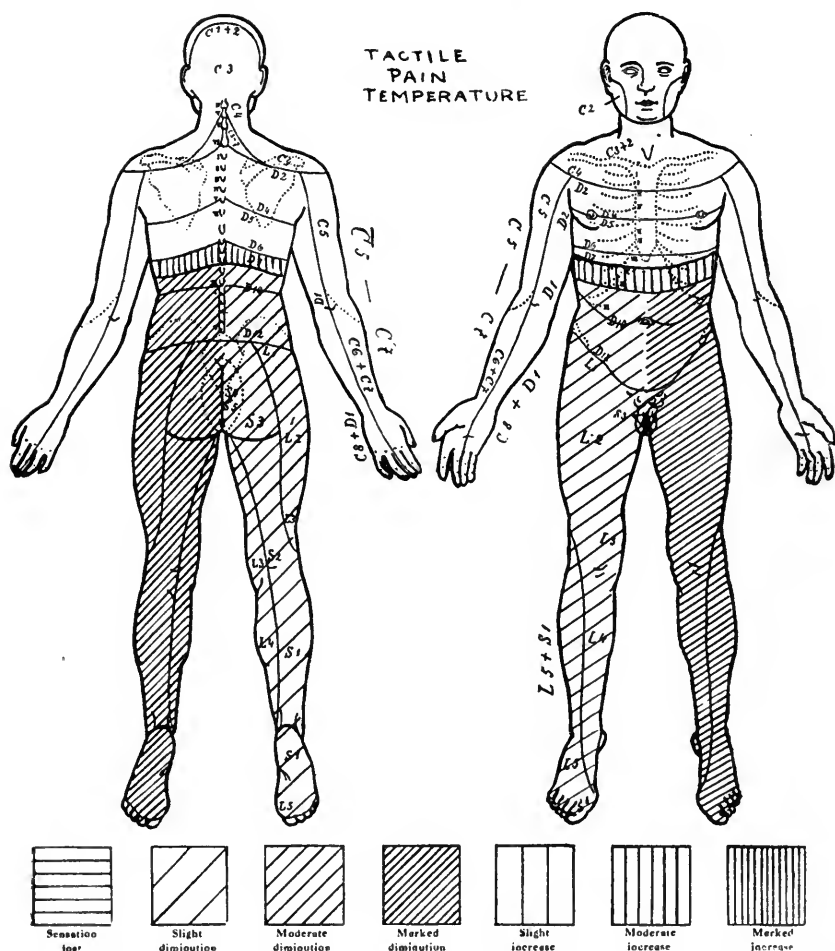


Fig. 1 (Mrs. L. G.).—False sensory localizing signs of a spinal cord tumor at the level of the eighth cervical segment. Compare with Figure 2.

corresponding area on the right side, there was a moderate loss of touch, pain and temperature sensibility. Above the level described there was a band of hyperesthesia for all sensations, about 2 inches wide. Behind, this hyperesthesia extended up to the spine of the eighth dorsal vertebra, which appeared to be tender to pressure. Sense of position of the toes and vibratory sense were completely lost in the right foot and leg; in the left toes, one mistake was made in five tests.

Roentgen-ray examination of the spine was negative. The fluid obtained by lumbar puncture was clear; globulin was not increased; there were no cells; the Wassermann test was negative.

Treatment and Course.—The diagnosis of an extramedullary tumor at or above the seventh thoracic segment was made and a laminectomy (fifth to ninth dorsal) was performed by me on June 26. The operation failed to reveal a tumor even after careful exploration, but it was noted in the record that the posterior spinal vessels were distended with blood and that, after the first escape of cerebrospinal fluid when the dura and arachnoid were incised, there was no further leakage of fluid from above. The patient recovered satisfactorily from the operation and she left the hospital against advice three weeks later. She was seen at her home about three months later. Her condition was unchanged.

Second Admittance to Hospital.—Nothing more was heard from her until March, 1915, about three and one-half years after the operation. She was then admitted into Mt. Sinai Hospital as a private patient of Dr. I. Strauss and was referred to me by Dr. Strauss for operative interference. During the three and one-half years the patient had become progressively worse; she had gradually lost all power in the lower extremities, and the upper limbs—especially that of the right side—had become weaker. She had lost complete control of the bladder, and her bowels had become obstinately constipated.

Examination.—Examination made March 11, 1915, revealed the following: The cranial nerves were normal, except that the right pupil was smaller than the left. Upper Extremities: Movements at the shoulders were good; at the elbows, flexion was good, extension weak, especially on the right side. The biceps reflexes were present and equal; triceps and radius and ulnar reflexes could not be obtained. Abdominal reflexes were not obtainable. There was a marked spastic paraplegia of both lower extremities. The patient was unable to move either limb and on the least irritation, there was a tremendous flexor spasm of both extremities. Knee reflexes were greatly exaggerated. Double ankle clonus, double Babinski and Gordon reflexes were present. There was a complete loss of all sensation—superficial and deep—below the level of the seventh cervical area on both sides (Fig. 2). The symptoms now pointed clearly to a tumor at the seventh to eighth cervical segments, on the posterior surface of the cord and to the right.

Treatment and Course.—At the operation (Dr. Elsberg) on March 15, 1915, the arches of the sixth and seventh cervical and first thoracic vertebrae were removed. When the dura was opened a tumor, about 1 inch long and lying on the posterior surface of the cord slightly more on the right side, was exposed (Fig. 3). The growth, which proved to be an endothelioma, was removed without difficulty, leaving a deep depression in the cord. Convalescence from the operation was uncomplicated, but there was no improvement in the paraplegia.

CASE 2.—History.—Spinal symptoms had been present for one year. There were symptoms at the eleventh to twelfth thoracic segments. Laminectomy was performed; no tumor was found. Seventeen months later, there were level symptoms at the fifth thoracic segment. Laminectomy was performed and an extramedullary tumor was removed at the fourth thoracic segment. The patient recovered.

H. R., 36 years of age, a patient of Dr. C. L. Dana, was admitted into the New York Neurological Institute in January, 1912. One year before, he first noticed a tickling sensation in the calf of the right leg and an annoying sensation as if the outer side of the leg were "rubbed with sand paper." A few months later he noticed that the lower part of his body was not as sensitive to cold as the upper. Three months later, the right knee had become stiff and he had difficulty in walking. Four months before admittance, the

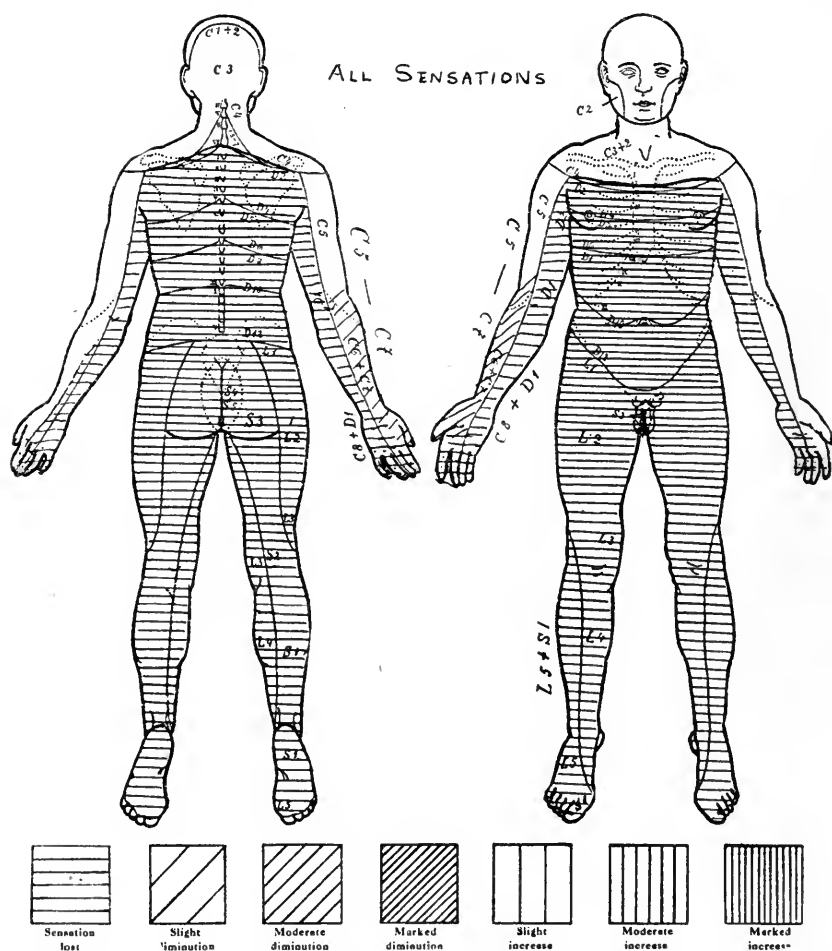


Fig. 2 (Mrs. L. G.).—Sensory disturbances, four years later. Correct sensory level signs of a tumor at the eighth cervical segment. Compare with Figures 1 and 3.

left lower extremity became weak and stiff. The weakness and stiffness of the lower limbs became progressively worse up to the time of admittance. He had no trouble with his upper extremities. For eight months he had increasing difficulty in urination.

Examination.—Jan. 25, 1912: The patient, who was strong and muscularly well developed, walked with great difficulty on account of marked spas-

ticity of both lower extremities. The cranial nerves were normal; nystagmus was not present; the pupils reacted well to light and accommodation. Upper Extremities: Power was good, that of the right equal to that of the left; the reflexes were active, those of the right equal to those of the left. The abdominal reflexes were active, those of the right side equal to those of the left. Cremasteric reflexes were weak on both sides. Lower Extremities: Both were spastic; the knee and ankle reflexes were much exaggerated. Double ankle clonus and the double Babinski reflex were present. There was no disturbance of the articular or vibratory sense. There was no disturbance of tactile sense, the slightest touch with absorbent cotton being felt distinctly over both lower limbs. Heat and cold were not felt below the level of the eleventh thoracic segment; pain sensibility was distinctly diminished over the third and fourth lumbar segments on each side, and slightly diminished on L 2. Just below the umbilicus, there was a band of marked hypalgesia about 2 inches

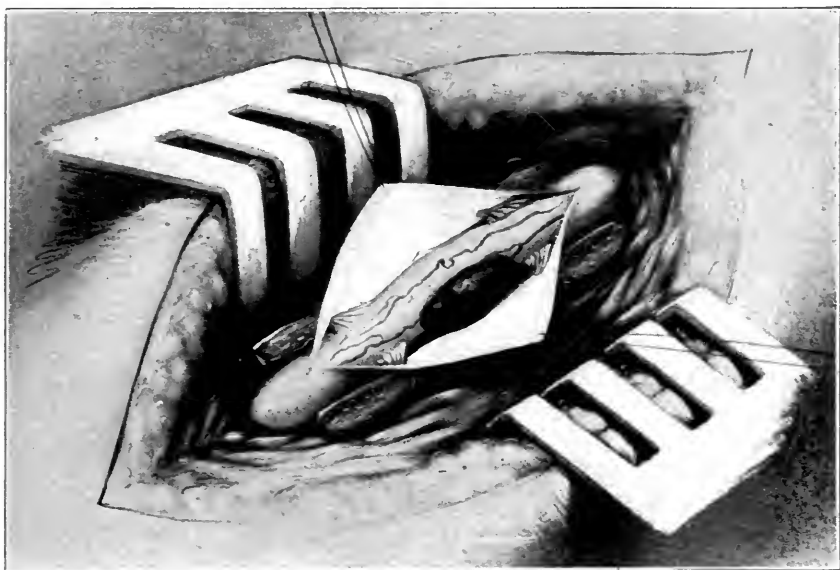


Fig. 3 (Mrs. L. G.).—Extramedullary tumor at the eighth cervical segment. Compare with Figures 1 and 2.

wide, which extended all around the body (Figs. 4 and 5). The Wassermann tests of the blood and cerebrospinal fluid were negative. The roentgen-ray examination was negative.

The patient left the hospital after a few weeks; he returned four months later in a much worse condition. His back had become stiff; he was bed-ridden. The lower limbs were much more weak and stiff. He was having considerable bladder trouble, and he could not control his bowel movements.

Second Examination.—Physical examination, July 4, 1912, revealed: The upper extremities were normal. The upper abdominal reflexes were present and active; the lower abdominal reflexes were weak. The spasticity of the lower limbs had increased very much, and there was an almost continual clonus of the extensors of the thigh. With great difficulty, the patient could lift his lower limbs a few inches from the bed. Flexor power at the knees was

weak. The knee and ankle reflexes were much exaggerated with double ankle clonus, Babinski and Oppenheim reflexes—a little more marked on the right side. There was no loss of vibratory or articular sense.

The sensory examination revealed: The disturbances extended on both sides up to the level of the eleventh and twelfth thoracic segments, and all sensations were involved, although pain and temperature sense were markedly affected, while tactile sensation was only slightly interfered with.

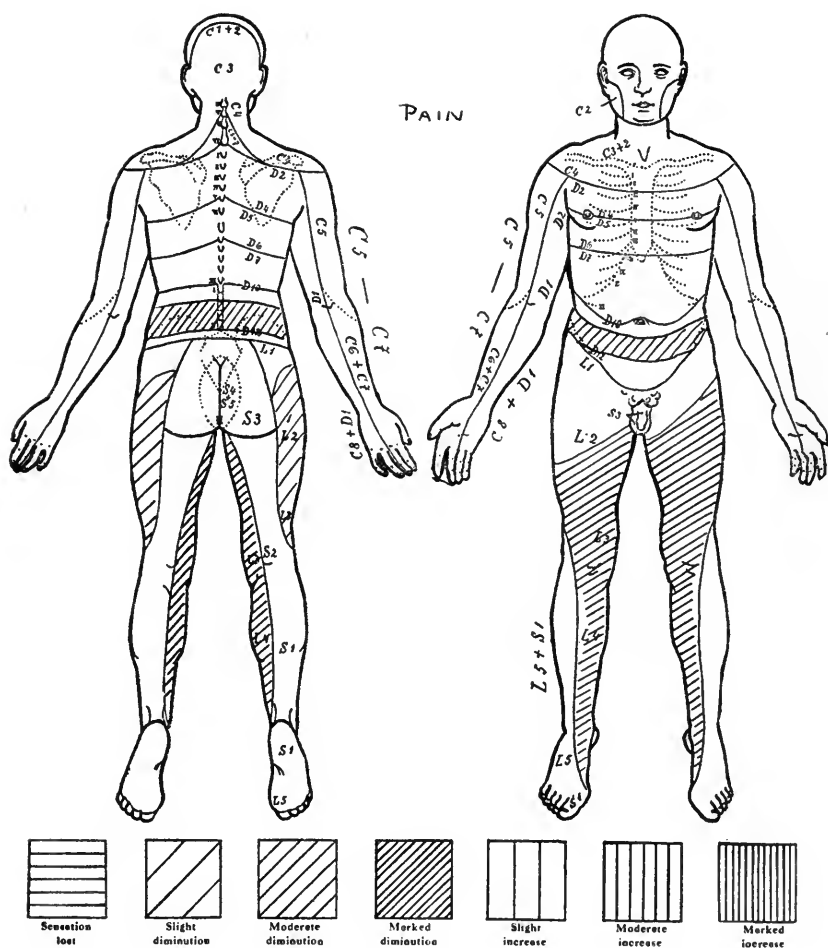


Fig. 4 (H. R., Jan. 25, 1912).—False sensory localizing signs of a spinal cord tumor at the level of the fourth thoracic segment. Compare with Figures 6 and 7.

Treatment and Course.—July 5, 1912, an exploratory laminectomy was performed by me. The arches of the ninth, tenth, eleventh and twelfth thoracic and of the first lumbar vertebrae were removed. When the dura and arachnoid were incised, the cerebrospinal fluid spurted out to a height of 12 inches. There was no evidence of tumor, the probe passed upward and downward in front

and behind the cord without meeting any obstruction. The wound was closed. The patient left the hospital unimproved after one month.

Second Admittance to Hospital.—Sixteen months later, he was seen at the Montefiore Home, where he had been admitted to the service of Dr. S. P. Goodhart, some months before. He had a paraplegia in extension, although with great effort he could flex his knees to about a right angle. The abdom-

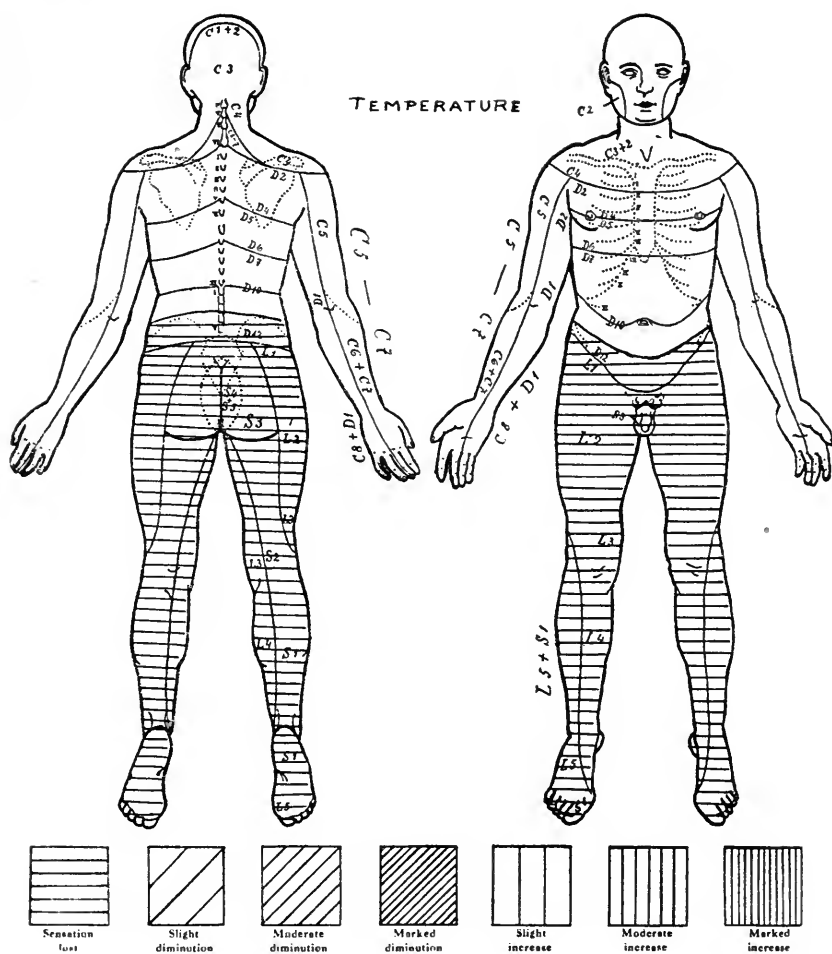


Fig. 5 (H. R., Jan. 25, 1912).—False sensory localizing signs of a spinal cord tumor at the level of the fourth thoracic segment. Compare with Figures 6 and 7.

inal and cremasteric reflexes were present but were weaker on the left. He had markedly exaggerated knee reflexes, double inexhaustible ankle clonus, double Babinski, Chaddock, Mende and Gordon reflexes. There was a well marked loss of articular sense in the left lower limb. The sensory changes (Figs. 6 and 7) extended up to the fifth and sixth thoracic segments.

Treatment and Course.—Jan. 2, 1914: Laminectomy was performed by me. Arches were removed at D 4 and 5, and then arches at D 2 and 3. The extramedullary fibroma which lay behind and on the left side of the cord and pushed the cord over to the right (Fig. 8) was removed. The tumor was easily raised from its bed and removed. It measured 3 by 1.5 cm. The patient improved rapidly and six months after the operation was entirely well.

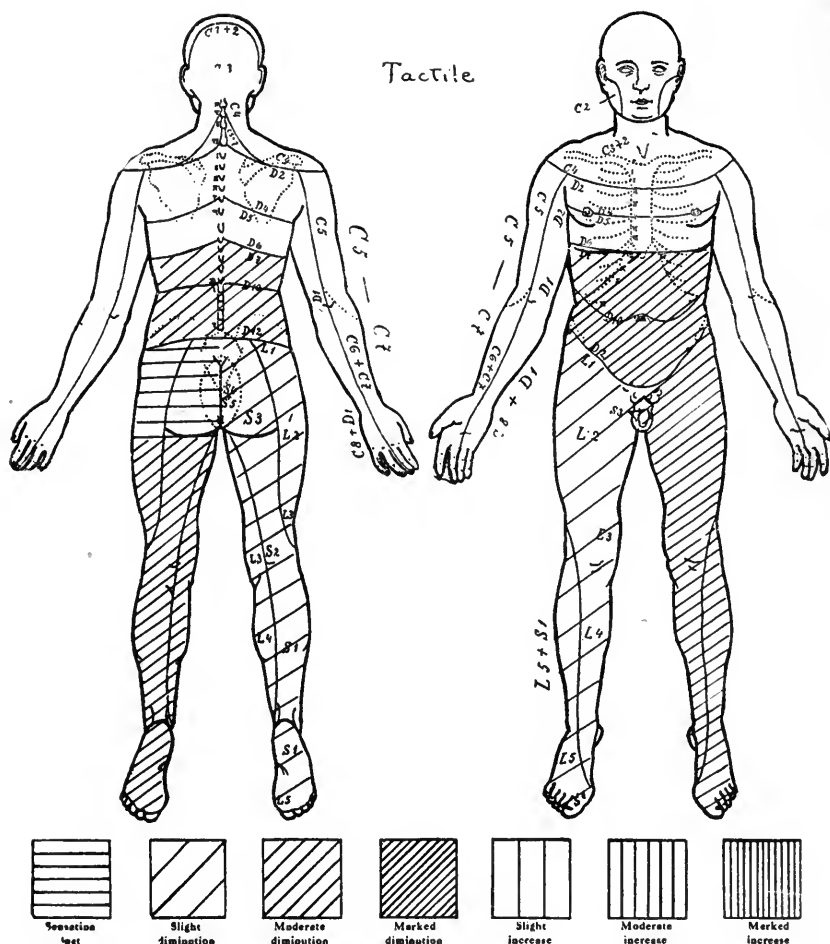


Fig. 6 (H. R., Nov. 29, 1913).—Sensory disturbances seventeen months later than those shown in Figures 4 and 5. Correct sensory signs of a tumor at the fourth to fifth thoracic segments. Compare with Figures 4, 5 and 8.

To summarize: In Case 1 there were at first distinct level signs at the seventh thoracic segment and three and one-half years later, signs at the eighth cervical level. The tumor was removed from the eighth cervical level. In Case 2 there were signs of a tumor at the fourth thoracic level; two years before there were signs and symptoms of a new growth at the eleventh thoracic segment. In each of the

patients, the first laminectomy was performed at a much lower level than that at which the tumor was found at the second operation.

How can these cases be explained? It will be noted that, in both instances, there were no root symptoms, and that in Case 1, the symptoms referable to the upper extremity appeared several years after those referable to the lower limbs.

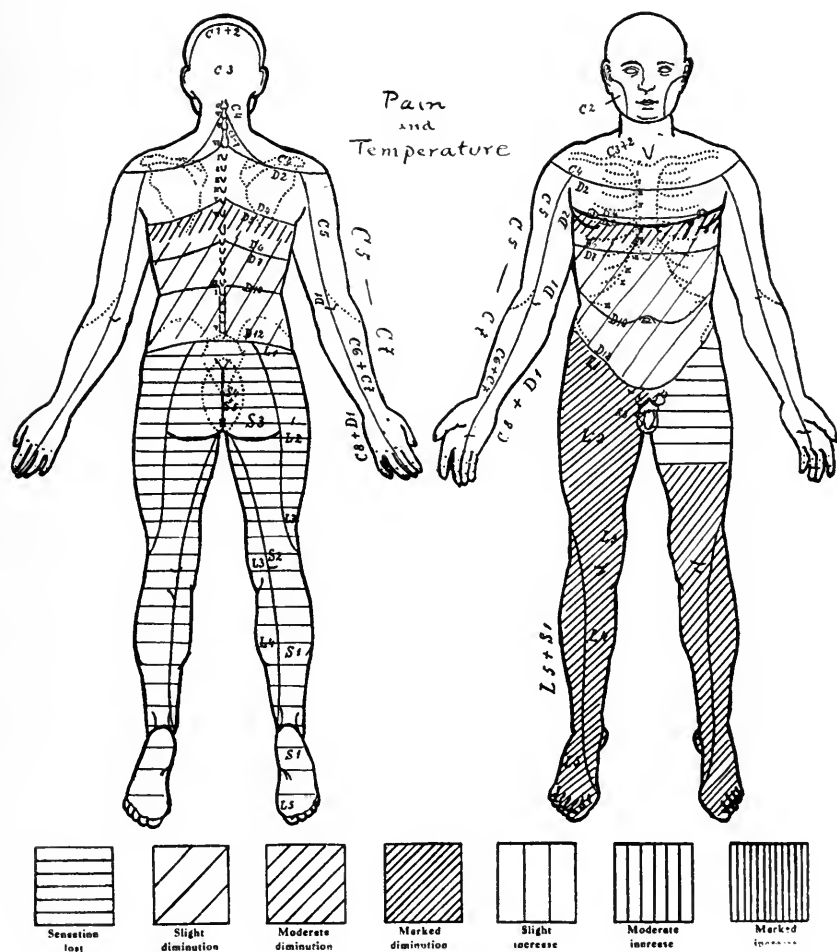


Fig. 7 (H. R., Nov. 29, 1913).—Sensory disturbances seventeen months later than those shown in Figures 4 and 5. Correct sensory signs of a tumor at the fourth to fifth thoracic segments. Compare with Figures 4, 5 and 8.

In looking over my records, I have been impressed by the frequency with which patients with spinal tumors in the cervical region first complain of sensory and motor symptoms referable to the lower extremities. In twenty-five cases of extra- and intramedullary cord tumors in the cervical region, six patients stated that the first symp-

toms they observed were in the lower extremities; in three, weakness, stiffness and sensory disturbances preceded symptoms in the upper limbs by many months, and two of the five patients had no knowledge of any motor or sensory disturbances in their upper extremities when they were first examined.

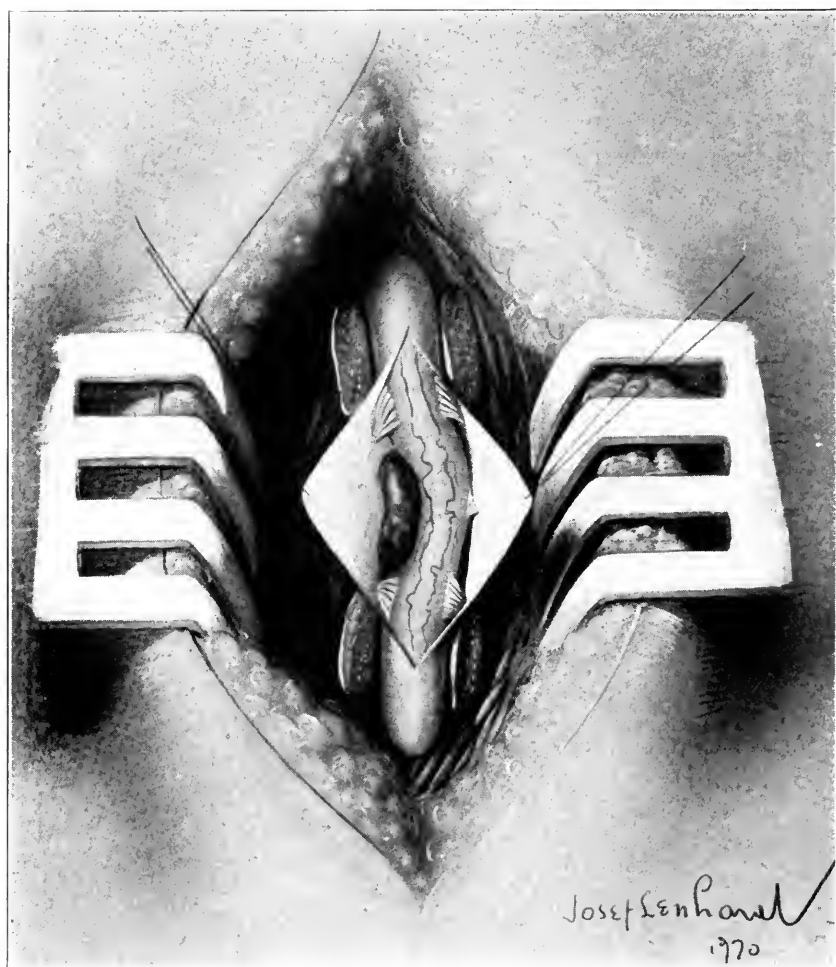


Fig. 8 (H. R.).—Extramedullary tumor at the fourth thoracic segment. Compare with Figures 4, 5, 6 and 7.

When, in an expanding intramedullary or extramedullary spinal lesion, the loss of sensation is not a complete one, the boundary between parts of normal and abnormal sensibility is often uncertain. If the impulses which pass upward have not been completely interrupted at a certain level, the area of disturbed sensation may merge

gradually into parts of normal sensation. This change from disturbed to normal may be so gradual that above the area in which sensibility disturbances can be recognized with the ordinary tests, there may be a broad area (extending over a number of cord segments) in which sensation is apparently normal. As soon as the interruption becomes more marked, however, the sensory disturbances shift to a higher level, which is the true segmentary level.

It is probable that the fibers for the different extremities and even for parts of the extremities are grouped together in the spinal pathways.³ If there is such a definite grouping of sensory fibers, one can understand that with the gradual crossing of the fibers for pain and temperature, and perhaps also for tactile sensation, only a small part of these fibers may at first be interfered with. The affected fibers may be those that supply areas considerably below the actual level of the lesion, and hence the sensory level obtained early in the course of the disease may be one considerably below the real level of the lesion.⁴

ERRORS IN INTERPRETATION OF SIGNS REFERABLE TO THE SIDE AND
PART OF THE CORD AFFECTED

In a paper published some years ago,⁵ attention was called to the fact that, in rare instances, a tumor on one side of the cord may dislocate the cord to the opposite side to such an extent that the pressure of the cord against the wall of the spinal canal may cause symptoms referable to the side of the cord opposite to that of the tumor. I have seen two cases of this condition, in which, with ill-defined Brown Séquard symptoms, the greatest motor loss was on the opposite side and the marked sensory disturbances on the same side as the tumor. In both these patients the tumor had been localized on the opposite side to that at which it was found at operation. In a third patient, we were uncertain as to the side of the cord on which the tumor lay, because the marked motor symptoms and signs were on the opposite side, and the distinct sensory signs on the same side as the root symp-

3. Most of the evidence, hitherto adduced, seems to support the opposite point of view, i. e. that motor, and also to a great extent sensory, fibers are not grouped according to the areas they supply. In another paper I hope to give strong evidence that the contrary is true, and that there is a definite grouping of fibers within the sensory and motor pathways of the spinal cord in accordance with the parts of the body or of the extremities supplied.

4. Most writers state that the fibers conveying the sensations of pain and of heat and cold require from two to three segments for their crossing. I believe that Head is correct in his statement that the crossing is much more gradual, requiring from five to six segments.

5. *Am. J. M. Sc.* **149**:337 (March) 1915.

toms. The condition here described is analogous to what is not so rarely seen in tumors in the cerebellopontine angle. Paralysis of the seventh nerve may occur on one side with a tumor in the angle of the other side, because the tumor pushes the cerebellum over to the opposite side, compressing the facial nerve of that side against the petrous portion of the temporal bone.

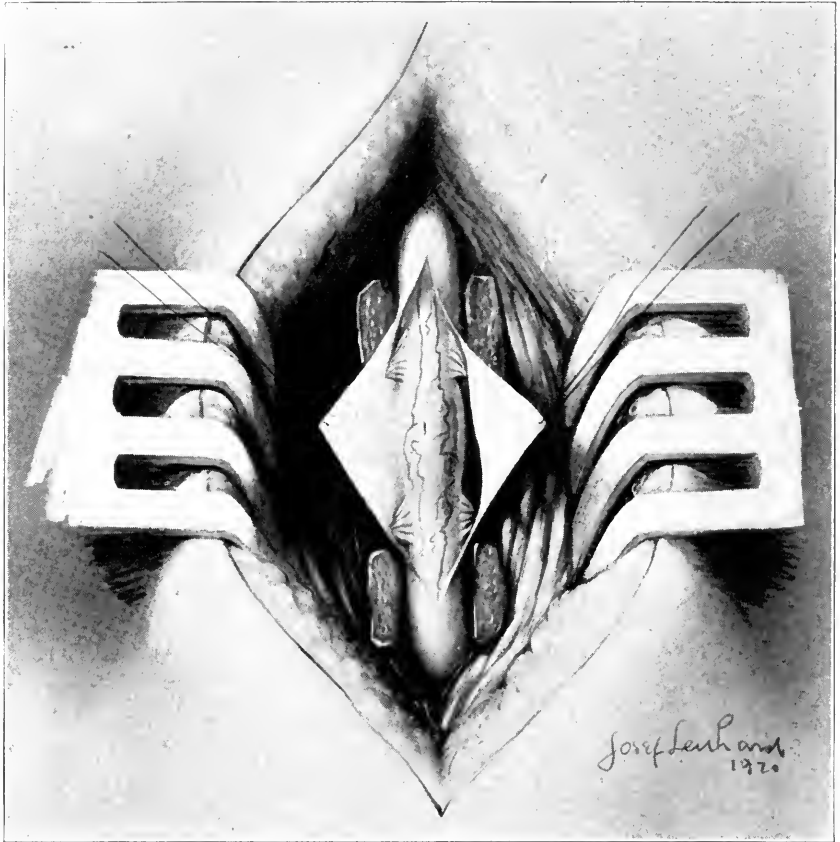


Fig. 9.—Intramedullary tumor in the posterior column. The growth is covered by a thin layer of cord tissue.

Especially in patients who give no history of root pains, the occurrence of more marked motor symptoms (and also, sometimes, of more distinct posterior column disturbances) on one side may mislead the examiner, so that the growth is localized on the wrong side of the cord. These cases are examples of reverse Brown Séquard symptoms and may occur when a tumor of soft consistency pushes a movable part of the cord to the opposite side.

It is possible also that in some patients with bilateral sensory and motor symptoms, the signs of interference with the tracts on the side of the tumor are due to direct pressure by the growth, and those on the other side are caused by the pressure of that side of the cord against the wall of the spinal canal. Such a mechanical condition would result in the kind of double Brown Séquard syndrome that has been described by some authors.

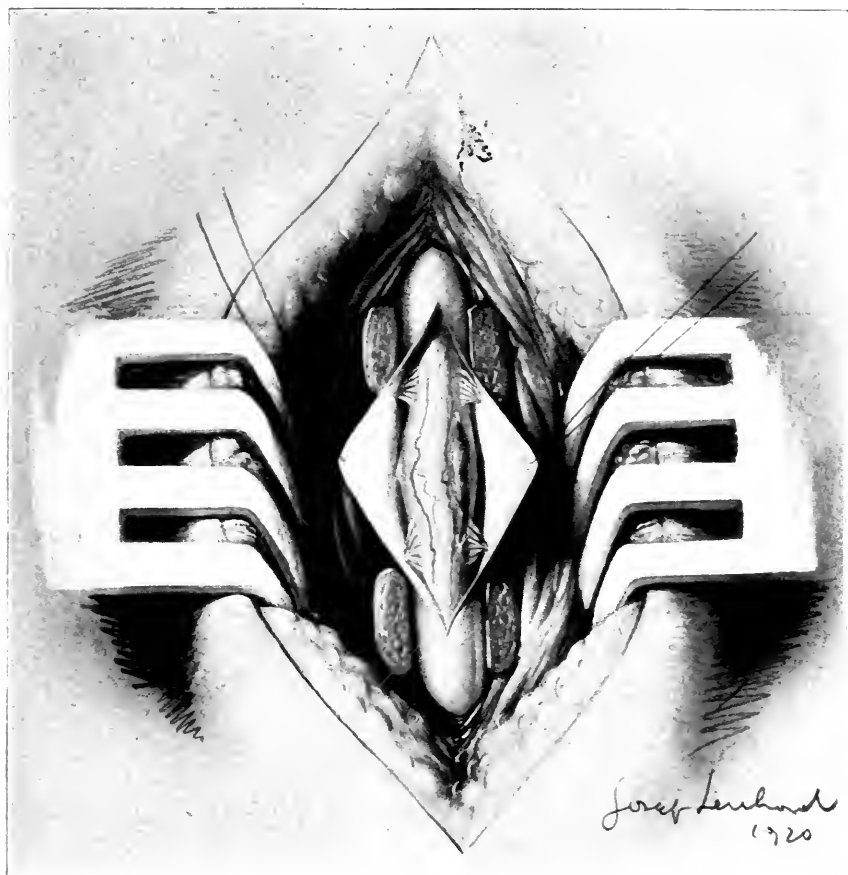


Fig. 10.—Partial extrusion of intramedullary tumor after an incision into the cord had been made.

As is well known, the fibers that convey the so-called deep sensations, the sense of position, of movement and of weight, the vibratory sense, and tactile discrimination, travel up the spinal cord in the posterior column of the same side. When these sensations are disturbed on only one side of the body, the diagnosis of a lesion on that side of the cord can be made with a fair degree of certainty. In many patients there is evidence of disturbance of both posterior

columns although one side is affected more than the other. In some patients, however, the signs of interference with both posterior columns are the same on both sides, and in such a case the question may arise whether the new growth lies on the posterior surface of the cord and is compressing both posterior columns, or whether the tumor is growing in front of the cord and is pushing the cord backward against the arches of the vertebrae. I have seen one case of this kind. In this patient I expected to find the tumor on the posterior surface of the cord on account of the well marked posterior column disturbances, but I was surprised to find that it lay in front of the cord.

Extramedullary tumors that grow on the anterior surface of the cord are often, as we have shown, difficult to differentiate from intramedullary growths. Root pains are rare and early muscle atrophies frequent.

Typical root pains may occur with intramedullary tumors, as may even unilateral posterior column disturbances, if the new growth within the cord substance is small and located in one posterior column near the origin of a posterior root.

Figures 9 and 10 illustrate this condition. The patient had root pains referred to the right side of the thorax and unilateral loss of vibratory and articular sense on the same side, with a spastic paraplegia and sensory level symptoms. The history was a typical one of extramedullary tumor, but at the operation a growth in the right posterior column within the cord was found. There is much to support the view that typical root pains can follow an affection within the cord near the origin of a posterior root.

PARESTHESIA IN ONE OR BOTH UPPER EXTREMITIES CAUSED BY TUMORS IN THE THORACIC CORD

In five cases of thoracic cord lesions of an expanding nature, the patients complained of tingling or hyperesthesia in the fingers of one or both hands. In three of these, an extramedullary tumor between the fourth and eighth dorsal segments was removed at the operation. In the first two patients we were much confused by this symptom, because it did not agree with the other level symptoms. In one of the patients, in fact, the spinal symptoms were supposed to be due to a diffuse lesion and the surgical interference was delayed for a considerable period on account of the symptoms in the upper limbs. One patient had been walking around on crutches, and we believed the tingling in the forearm and hands was due to pressure on the nerves in the axilla by the crutches. The other patients were bedridden, and in their cases some other explanation had to be found. I believe that the tingling in the fingers in these cases was due to a slight pres-

sure on, and irritation of, posterior roots by a column of cerebrospinal fluid above the tumor. In at least one of the patients, a large amount of fluid under great pressure escaped from above as soon as the tumor was removed. In all of the patients, the cutaneous hyperesthesia and the tingling had disappeared when the patient recovered from the anesthesia.

Abstracts from Current Literature

UEBER MYOKYMIE UND MUSKELVERÄNDERUNGEN BEI SKLERODERMIE (CONCERNING MYOKYMIA AND MUSCLE CHANGES IN SCLERODERMA). S. NEUMARK, Schweizer Arch. f. Neurol. u. Psychiat. 6:125, 1920.

The syndrome of myokymia was first described by Kny (1888) and Morvan (1890), the latter designating the condition as chorée fibrillaire. Schultz, in 1895, gave it the name it now bears. The cardinal symptom consists in localized, wavelike muscular contractions without motor effect. While there is a symptomatic myokymia which occurs in a large variety of organic diseases of the nervous system, the so-called essential type develops in persons who have previously enjoyed good health, but who have, in most instances, been subject to a slight infection; the condition disappears spontaneously.

Neumark reports a case presenting some unusual features. The patient was a man, 65 years of age, who had been seen the first time fifteen years previously, when a diagnosis of psychasthenia was made. Even then it was noted that the facies was mask-like; there was a constant wrinkling of the brow and fascicular contractions spreading over the entire face gave one the impression of slow, grimacing movements. The patient could bring these movements to rest, at will, for a short time, when the mask-like appearance became striking. A Chvostek II was noted; however, Trousseau's sign was absent; subsequently the patient developed attacks of unconsciousness. At the time of his examination, there was a tremor of the tongue and a fibrillary twitching along the edges, but no atrophy. Fascicular twitching was present over the entire face, but more marked on the left than on the right. There was still a continual wrinkling movement of the brow, which was aggravated by fatigue and by emotional disturbances. Aside from a sensation of tension there was no subjective sensory disturbance. In addition to the fibrillary tremor, there was a left-sided facial spasm. The mandible was somewhat atrophic; the scalp was thin, smooth, glistening and adherent; over the left mandible, a sclerodermic plaque was situated; and in addition, sclerodermic changes involved the hands and possibly the thigh. The hands were blue and cold and the lower extremities showed a marked cutis marmorata. There was a slight dorsal kyphosis. No sensory disturbances could be demonstrated objectively. The writer laid great stress on the fact that the movements about the face ceased entirely during sleep. Atrophic changes of the bones of the hands could be demonstrated by the roentgen ray. Aside from these symptoms, the examination was practically negative.

Neumark, in reviewing the reported cases of myokymia, found that the seat of the disturbance commonly involved the lower extremities, particularly the calves, although almost any muscle of the body might be involved. The etiologic factors given were overexertion, coryza, trauma, chronic lead poisoning, syphilis, tuberculosis, chlorosis and the neuropathic habitus. Men were principally affected; age ranged from 11 to 65, and no type of occupa-

tion was excepted. Muscular irritability was increased in most cases, as was also the excitability of the nerve trunks. The reflexes were usually increased. Vasomotor disturbances were common. The duration of the disease varied; in the majority of instances the movements disappeared in a few weeks, although they have been known to persist for twenty years, and in the case reported they had been present for more than ten years. The writer called attention to the fact that, so far as he is aware, no case of a myokymia complicated by scleroderma, has been described; various types of muscular atrophy associated with scleroderma are by no means uncommon. The part in the nervous system from which these movements arise is not known, although there is reason to believe that it may be situated in any part of the reflex-arc or even in the muscles themselves, as Oppenheim supposes. Kurschmann attributes the movements to neuritis. It is believed that there is a close relationship between myokymia and myotonia acquisita of Talma. Neumark thinks that the so-called essential myokymia is not dependent on an organic lesion of the nervous system, but is the expression of a functional disturbance.

In discussing scleroderma, the author quotes Hutchinson to the effect that associated muscular atrophy is the rule and is absent only in mild cases and in old people. The writer's patient was well along in years, which may explain the absence of muscular atrophy. The types of muscular disturbances noted in scleroderma may be classified as atrophy, myosclerosis and myositis, although none of these commonly occur alone; as a rule, there has been found a primary increase in the interstitial connective tissue with a resulting pressure atrophy of the muscle fibers, as well as a myositis. Insufficiency of the functions of the skin, compression of the muscles, blood vessels and nerves all contribute to the atrophy. Cases have been reported, however, in which the muscular atrophy preceded the cutaneous changes by several years and progressed independently of changes in the latter. Disturbances in the function of the skin, such as profuse perspiration, may also precede. Furthermore, myosclerosis may occur in a location of the body quite distant from the changes taking place in the skin. Myositis ossificans has been seen in association. It has been assumed that the muscular change of scleroderma may be the result of a general infection-intoxication process which begins as a myositis and ends in sclerosis. The involvement of the skin in this inflammatory process as well has been assumed and the entire process looked on as a dermatomyositis. Cassirer takes this standpoint for some of the cases and it is the one to which the writer adheres. A differential diagnosis between these and the other types of scleroderma may therefore be impossible. The histopathologic changes noted show an atrophy of the muscle parenchyma, peri-arteritis and endarteritis, endophlebitis, perimuscular and intramuscular sclerosis, disappearance of the cross-striations, splitting of the muscle fibers, homogenization of the protoplasm, and small round cell infiltration. While the reports of different investigators differ in some respects, they all seem to emphasize the importance of vascular changes in bringing about the disintegration of muscle. It is true that the former may in turn be due to some lesion of the nervous system. The writer is unable to say what relation, if any, the myokymia in his case bore to the scleroderma; he is inclined to think, however, that they are two separate and distinct entities, although they may both be manifestations of one and the same general neurosis.

UEBER DIE PSYCOPATHISCHE KONSTITUTION BEI KRIEGS-NEUROSEN (THE PSYCHOPATHIC CONSTITUTION IN RELATION TO WAR NEUROSES). FRITZ FRÄNKEL, *Monatschr. f. Psychiat. u. Neurol.* 47:287 (May) 1920.

The majority of investigators believe that a constitutional predisposition is an important factor in the development of war neuroses. The author analyzes seventy-two cases of war neurosis with this point in view.

The concept of "psycopath" lacks any exact objective criteria. The stigmas of degeneration are valueless, and our judgment must be based on the past history of the patient. The demarcation between normal and psycopath is gradual and, in a sense, arbitrary. Arbitrary also is the grouping of the psycopathic states, for there are all grades and all transitions. The author adopts the following classification: imbeciles, 8; unstable (*haltlose*), 5; unbalanced (*verschobene*), 3; constitutionally irritable, 14; constitutional neurasthenics, 10; sensitive, 19, and cyclothymic and depressive, 2. The different classes are discussed at some length.

The unstable (*haltlose*) are characterized by the instability of their lives. One of this group held twenty-four positions in a short time. Many of this group, 54 per cent. according to Kraepelin's statistics, come into conflict with the law. This class includes many pathologic swindlers and pathologic liars.

The unbalanced (*verschobene*) are characterized by a sense of superiority and a heightened sense of self regard. They feel superior to their station in life and are always reaching out for higher levels both socially and intellectually. They are unstable in their relations to the opposite sex.

The constitutionally irritable (*konstitutionel erregte*) form the second largest group. They lack the normal inhibitions and self restraint, fly off the handle, fight at the drop of the hat and are given to outbursts of violence. In civil life these people have a chance to relieve the accumulating tension by emotional outbursts, but the strict army discipline prevents this and causes increased emotional repression, so that they seek the only way out—escape in sickness. In no class of psycopath is the character of the war neurosis as defense reaction more evident than in this class. It includes the most primitive reaction types whose protest character is grossly demonstrated in a paroxysm of striking, kicking and shrieking or in blocking of consciousness in unconscious spells or dream states.

Characteristic of constitutional neurasthenics are hypochondriacal complaints of all types. They suffer from every conceivable sort of bodily weakness. Objectively, we find vasomotor instability. It is uncommon for this group to stand any long period of field service.

The sensitive group includes timid, dependent persons, abnormally sensitive to any threat of physical violence. They resemble the neurasthenics in the tendency to vasomotor instability. Their psycopathic trend shows itself in early childhood. They cannot see any one hurt, cannot endure see-saws and merry-go-rounds, and are subject to night terrors and anxiety dreams. Later, as adults, they are timid in the presence of strangers and easily embarrassed. They are seclusive and have few friends. They are shy in the presence of women, and apt to be sexually abstinent until marriage. Alcohol intolerance is common. This type includes the largest number of cases.

Summarizing, the author states that the endogenous factor—the psycopathic constitution—is most important in determining the causation of war neuroses. Exogenous factors are much less significant. Sixty-one of the seventy-two cases show this clearly in the past history. Of the eleven others, whose his-

tory was not so definite, there was not one who presented a normal personality. According to Gaupp, even if the family history shows no evidence of mental or nervous disorder, and the patient states that he has always been well, the mere fact that he developed symptoms in a situation which left his comrades unaffected, demonstrates that the essential cause of his trouble lies within himself. The objection may be raised that this makes the concept of the *psycopathic* constitution too broad. But the fact is that the great experiment of the war proved that the number of abnormal personalities was vastly greater than we formerly believed. The war brought them out. There is danger in this, because the laity has come to feel that groups of nervous symptoms are the natural result of mechanical and psychic traumas. We may have a mass development of such states as traumatic neuroses. It behooves us as physicians to educate people in the belief that we are capable of standing even exceptional traumas without developing symptoms, and that when symptoms do develop under strain they result from our own weakness rather than from the trauma itself.

SELLING, Portland, Ore.

RUPTURE OF THE SPINAL CORD IN DYSTOCIA. F. H. KOOY,
J. Nerv. & Ment. Dis. 52:1 (July) 1920.

The patient whose case is reported was the fifth child. The first two children were in transverse position when labor began; the third and fourth were in normal position. The patient was also in transverse position in utero, with the head offering some difficulties, and was revived with difficulty after birth. Three days later, ulcers appeared on the buttock which rapidly increased in size. The child could never move its lower legs, feet or toes; there was only slight flexion at the hip. At 1½ years, the child burned its legs without pain. It had always had incontinence of urine and feces. At the age of 2 the legs were paretic and spastic, with slight movement at the hip. Thermic sense was lost in the legs and gluteal region: there was a large, deep bed sore on the sacrum. While in the hospital, he suffered a spontaneous fracture of the femur. At the age of 7, the paralysis became atonic with loss of Babinski sign, with knee jerks diminished and ankle jerks absent. Touch was lost in the lower part of the legs and diminished in the upper part of the legs. An abscess over Poupart's ligament was incised without pain; the decubitus was increased in size; incontinence of urine and feces continued. At the age of 8, knee jerks were lost. The patient died at the age of 9. The diagnosis was lesion of the cord resulting from dystocia.

Postmortem examination demonstrated that the decubitus had penetrated the sacrum; there was no abnormality of the vertebral column. At about the seventh thoracic vertebra the cord was found to be a thick, solid ring of fibrous tissue with adherence of the dura. Practically the entire cord was examined in serial sections, alternately stained with Van Gieson's and Weigert-Pal's methods. At the level of the lesion the structure of the cord was unrecognizable. There was a network of connective tissue with small islands of glia and a small bundle of nerve fibers which when traced down proved to be the direct pyramidal tract. On this finding the author explains the small amount of movement at the hip. The ascending and descending degenerations of the cord were very painstakingly worked out. Marchi's stain, of course, could give no information since the case was of too long standing but with the Weigert method the author traced the various tracts.

He classifies lesions of the spinal cord dependent on dystocia as occurring in three different ways: (1) by venous engorgement, compared to hemorrhages in the adult in states of sudden venous congestion from vigorous movements, etc. He states that published reports of cases in which venous congestion is probably the only cause of these hemorrhages in the spinal cord of the newborn are not many, and that multiple hemorrhages are commonly found; (2) by fracture, rupture, or luxation of the vertebral column and a secondary meningeal and medullary hemorrhage with more or less complete compression of the cord; (3) partial or complete rupture of the cord, with the vertebral column intact. But he also adds that it is impossible to tell by microscopic examination at all times the precise mechanism of its origin, and most difficult of all is the differentiation of a rupture of the cord from hemorrhage, as a large hemorrhage may cause a complete destruction of the spinal cord.

The author discusses the question of the reflexes (Bastian's law) but does not quote any of the modern war literature on the subject. The cause of the patient's decubitus is rather interesting since it appeared the first few days after birth and pressure could not have been responsible. The author decides in favor of trophic influences. He also discusses the fact that although the child could flex both hips, only one anterior pyramidal tract was intact, and he reaches the conclusion that each ventral pyramidal tract serves as a path for bilateral innervation of the legs.

WINKELMAN, Philadelphia.

THE RESULTS OF SECONDARY SUTURE OF PERIPHERAL NERVES.

JOHN S. B. STOFFORD, *Brain* 43:1 (May) 1920.

In this article are reported the preliminary results of secondary suture of nerves in 271 cases. The author systematically discusses the general factors which influence the progress and results, the technic of repair, conditions found at operation, complications and the comparative results of suture of the different nerves.

The delays to operations that are encountered after primary wounds are mainly sepsis, and in four cases ununited fractures. A period of from twelve to eighteen months' delay in suturing does not necessarily offer an obstacle to regeneration; but sutures in proximal parts offer decidedly better prognosis, and the larger the nerve trunks, the greater the amount of regeneration. The best operative technic is freeing the nerve ends, with wide resection to normal nerve tissue, suture without torsion through the neural sheath and with one through-and-through tension suture. Embedding the nerve in a new place, preferably in healthy muscle, insures against secondary scar formation and subsequent compression. Wide resection of ends in incomplete divisions is indicated to gain the maximal regeneration, as it has been shown that greater contractures and mechanical disabilities are associated with incomplete divisions.

In the presence of sepsis, intraneural changes take place extensively, even so much as 8 inches above the point of injury. Ligature of the main artery in the proximal part of the limb is mentioned as an aid to restoration, only to be condemned. In ununited fractures, the bone condition should be repaired first and after this has healed, the nerve can be sutured with much better chances of success.

As to results in secondary suture, none are perfect; there is always a deficit, and in this series, sensory regenerations were particularly disappointing. As to factors which hinder complete recovery: Intraneural changes in the proximal end, obstruction to the downgrowth of new nerve fibrils, destruction

of nerve branches and "bad shunting" are cited. Further, the loss of afferent stimuli from joints, muscles and tendons accounts for the disability of the limb, for whereas the individual muscles show return of power and the superficial sensation is fairly completely restored, yet the patient is unable to use the limb well except when he is watching it, and has lost those vital necessities for definitive use—the sense of position and appreciation of movement.

Suture of the musculospiral nerve gave uniformly good results, particularly when the injury was high up. The nearer to the cord, the more complete and rapid the return of function. Loss of synergic action in the extensors of the muscles accounts for the weak hand grasp and poor recovery that are generally encountered in the thumb extensors.

In the median nerve suture, an incomplete recovery of protopathic sensibility was general, and recovery of the voluntary power in the abductor brevis pollicis occurred in but 40 per cent.

The ulnar nerve recoveries were disappointing, possibly from "bad shunting" and from the small nerve supply to the intrinsic muscles of the hand, rendering the chances greater of their not receiving efferent fibers. Brouwer's hypothesis that the vulnerability of these finer muscles and nerves is due to the "finer construction of the thumb and higher functional significance which it has received in the phylogenesis" is offered as a suggestion in explanation.

In the sciatic, return of function in the tibialis posticus and flexors of the toes was poor. The external popliteal failures are accounted for by the inability to procure a good bed after suture, and the tendency to involvement of the nerve in scar tissue. In the internal popliteal, functional results obtained were good except in the restoration of sensation. End-to-end suture was done in all cases, and no nerve grafting or splitting operations were performed.

PATTEN, Philadelphia.

VISUAL FIELD FINDINGS IN A CASE OF BRAIN TUMOR. WALTER R. PARKER, *Am. J. Ophth.* **3**:736 (Oct.) 1920.

J. M., aged 28, first seen in 1911, a sailor, was sent home on account of inattention and inability to keep awake. Four years before he suffered attacks of headaches. About two years later headache increased and was often accompanied by nausea and vomiting, with occasional brief attacks of vertigo and falling sensation. He never complained of loss of vision. In 1911 vision was: right eye, 6/15; left, 6/12. Pupil reaction was present but was somewhat sluggish, especially in the right eye. Wernicke's sign was present. There was edema of both disks, most marked in the right eye, but there was no measurable swelling.

The headaches did not increase in severity, but mental and motor disturbance gradually became more marked. When he developed symptoms of hyperpituitarism a sellar decompression operation (transsphenoidal route) was performed by Dr. R. B. Canfield. Shortly after this symptoms of hypopituitarism appeared.

The patient died April, 1914. Postmortem examination showed a tumor involving the right tract, chiasm and adjacent brain substance. Pathologic Report: Large round celled sarcoma with numerous calcareous concretions (psammoma); growth probably primary in the meninges.

The visual fields showed first a homonymous hemianopsia without involvement of the muscular region. Vision in the right eye was 6/15; in the left eye, 6/12. Later the macula became involved and vision in the right eye was

1/60; in the left eye, 5/30. Still later there was a loss of green perception in the right eye with a marked contraction of the temporal form field. This was followed by loss of all color sense in the right eye with marked temporal contraction for form. Vision: Fingers could be seen at a distance of 2 feet. Finally there was total blindness in the right eye.

Both optic disks gradually paled and became atrophic. Examination by Dr. C. D. Camp, March 11, 1912, revealed general muscular weakness, slight difficulty in turning to the right, no nystagmus, power of convergence normal, slight facial paralysis, a positive Romberg sign, the knee reflex markedly exaggerated and a slight memory defect. The Wassermann and urine tests were negative.

Discussion.—Apparently the tumor first involved the right tract inducing a left homonymous hemianopsia and a positive Wernicke's sign. Later it either invaded the chiasm or led to pressure symptoms that affected only the papillomacular bundle causing loss of central vision. Finally a possible bitemporal hemianopsia developed, and this superimposed on a homonymous hemianopsia led to blindness in one eye (right) and loss of the temporal field in the other.

REESE, Philadelphia.

EPIDEMIC ENCEPHALITIS: INCLUDING A REVIEW OF ONE
HUNDRED AND FIFTEEN AMERICAN CASES. ARTHUR D. DUNN
and FRANCIS W. HEAGEY, *Am. J. M. Sc.* **160**:568 (Oct.) 1920.

The authors have carefully analyzed 115 cases of epidemic encephalitis. The frequent incidence of respiratory infections (31 per cent. occurring in close relationship to the development of encephalitic symptoms) is a point of etiologic interest. The various forms described, polio-encephalitic, lethargic, parkinsonian, cataleptic, meningitic, cerebral, polyneuritic and myelitic, must not be regarded as clear-cut clinical types. Not only is there considerable overlapping, but the symptomatic complexion of the individual case frequently changes during the course of the disease. Lethargy was noted in 79 instances, third nerve palsies in 66, diplopia in 58, headache in 57, fever in 56 and sixth nerve palsies in forty. The occurrence of catalepsy and catatonia in twenty-six cases is in line with the experience of other observers, and is an addition to the literature on this subject. In the face of the incidence of catatonia in the greatest diversity of conditions, not only in the psychoses and psychoneuroses, but also in organic brain diseases and infectious diseases, it is difficult to understand why it is still so often regarded as synonymous with dementia praecox and further why it is often given a purely psychologic interpretation.

The serologic analyses demonstrated that in twenty-five counts on fifteen cases there was an average leukocytosis of 10,200 with a neutrophilic percentage of 71; in sixty-four spinal fluid examinations there was an average of sixteen cells per c.mm., chiefly of the mononuclear variety with a positive globulin test in 50 per cent. of the cases and a mild syphilitic gold chlorid reaction in seven of eleven fluids. At present we are restricted to the following pathologic picture: (1) meningeal edema and thickening; (2) softening and congestion of both gray and white matter of the brain and pituitary gland; (3) punctate hemorrhage in mesencephalon and thalamus and basal ganglions; (4) thrombosis of small vessels; (5) perivascular infiltration of small vessels of the brain stem; (6) edema of the mesencephalic area.

The meager extent of our etiologic knowledge is reflected in the far from favorable prognosis. In 100 cases reported in the American literature, thirty-one patients died. The treatment is necessarily in the symptomatic stage. Lumbar puncture is of decided benefit.

STRECKER, Philadelphia.

FORMS OF PERIPHERAL NEURITIS AMONG TROOPS SERVING
—WITH THE EGYPTIAN EXPEDITIONARY FORCE. 1915-1919.

F. M. R. WALSH, *Brain* **43**:74 (May) 1920.

The commonest organic nervous diseases encountered in this campaign were multiple neuritis and the postdiphtheritic paralyses. No cases were seen due to malaria or dysentery, nor were there any instances of acute febrile polyneuritis. The total number was 160, one group of which followed faucial diphtheria, the other followed diphtheritic infection of skin wounds.

Of the faucial type, patients developed palatal paralysis in the second or third weeks of illness. Diminution or abolition of knee reflexes were not initial symptoms. Subjectively, aching pains in the legs on exertion, painful cramps in muscles, numbness of the feet, tenderness of muscles, and lively tendon jerks were the premonitory manifestations. The tendon jerks, however, usually diminished later.

In the 1916-1917 epidemic of diphtheria, the neurologic involvement reached a high percentage because of small dosages of antitoxin, and the premature return of patients to physical activity.

Sixty cases of multiple neuritis followed septic skin "sores," in several of which the diphtheria bacillus was isolated. The relation of the condition to diphtheria was practically certain; the patients came from areas where diphtheria was prevalent, and all gave histories of sores on the exposed skin surfaces which yielded slowly to treatment. The appearance of signs and symptoms in these cases followed a fairly definite order. There was first local paresis in the region of the site of the infective focus; then two to three weeks later paralysis of accommodation, and still later polyneuritis. Loss of accommodation was present in but one third of the cases; but infective foci, multiple usually, and polyneuritis were common to them all.

The author believes, in the light of the anesthesia and paralysis about the focus of skin infection, that the palatal paralysis in faucial diphtheria is a true local palsy. Consequently, despite the location of diphtheritic infection, the symptoms are clinically the same. This leads to the hypothesis that the toxins are conveyed by the perineural lymph channels, and therefore the nerves nearest the focus of infection are the first to become paralyzed through the involvement of those segments in the central system. Selective action is pointed out as a probable explanation of the paralysis of accommodation and a blood-borne toxemia causing the polyneuritis.

Of interest is the resemblance of this condition, following the above hypothesis, to tetanus.

PATTEN, Philadelphia.

TRAUMATIC REFLEX IMMOBILITY OF THE PUPIL. B. FLEISCHER
and E. NIESENHOLD, *Klin. Med. f. Augenh.* **64**:109 (Jan.) 1920.

After a review of the literature, a case is described in an otherwise healthy girl, 17 years of age, seen six days after her left lower lid was struck by a hay fork. The oculomotor nerve was completely paralyzed; the pupil was wide without reaction; abducens and trochlearis were functioning; the disk was not sharply defined; the surrounding retina was opaque; vision was 2/12.

The other eye was normal. The condition improved to a certain extent. After thirteen months the temporal portion of the disk was pale; vision, 1. The field was normal; there was some diplopia.

There was no reflex immobility, but an incomplete absolute unilateral immobility with almost complete abolition of direct and diminished consensual reaction to light; there were impaired accommodation and partial persistent paralysis of exterior muscles, caused by an orbital injury, similar to Laquer's case. The consensual, although incomplete, reaction with almost entirely lacking direct reaction, is attributed to the partial optic atrophy, inhibiting the conveyance of the lighter stimulus, and not able to arouse the function in the damaged light reaction fibers of the oculomotor nerve. The prompt consensual reaction of the other pupil proved that the transmission of the light stimulus in the optic nerve was not suspended.

The authors are tempted to assume in this case that a special damage to the pupillary fibers of the optic nerve occurred. They believe that also in the other traumatic cases the seat of the disturbance must be sought in the periphery, mostly in the orbit. On account of the almost missing direct, and insufficient consensual, reactions the authors assume a rather feeble impulse of convergence, through the existence of special convergence fibers, which escaped the injury, damaging only the light reaction fibers. The lesion might have damaged the ciliary ganglion, so that the finer transfer of the stimulus in the ganglion was rendered difficult or impossible; but the fibers penetrating or passing by the ganglion, namely, the supposed convergence fibers, were not affected.

REESE, Philadelphia.

THE DEVELOPMENT OF THE SYMPATHETIC NERVOUS SYSTEM IN MAN. ALBERT KUNTZ, *J. Comp. Neurol.* **32**:173 (Oct. 15) 1920.

In a series of about ten publications, Kuntz has reviewed the development of the sympathetic nervous system in representatives of the leading groups of vertebrates, finding in all cases certain broad similarities. The primordia of the sympathetic trunks and the prevertebral plexuses arise from cells of cerebrospinal origin which advance peripherally along both the dorsal and ventral roots of the spinal nerves. The vagal sympathetic plexuses, namely, the pulmonary, the cardiac and the enteric plexuses, except in the aboral portions of the digestive tube, arise from cells of cerebrospinal origin which advance peripherally along the vagi. In the more distal portions of the digestive tube the enteric plexuses arise from cells which are derived from the sympathetic supply in the lower trunk region. The ganglions of the head sympathetic of mammals were studied chiefly on the pig.

These results have been in part confirmed and in part adversely criticized by later students of the subject, the account of the origin of the cranial sympathetic ganglions having been especially attacked by Stewart (*J. Comp. Neurol.* **31**: 1920), who studied the rat.

Kuntz has now restudied the entire question on the basis of an examination of the extensive series of human embryos in the Mall collection. Regarding the controversial points in the development of the cranial sympathetic ganglions, he confirms most of his earlier observations made on the pig and at the same time admits the correctness of Stewart's findings. This implies that most of the ganglions of the head sympathetic nerves have a double origin, only one component of which was recognized in the author's earlier studies. For the details we must refer to the original text, which is accompanied by numerous photographs of the preparations.

HERRICK, Chicago.

ZUR HETEROTOPIE DER PLEXUS CHOROIDEI (CONCERNING HETEROTOPIA OF THE CHOROID PLEXUSES). S. KITABAYASHI, Schweizer Arch. f. Neurol. u. Psychiat. 6:154, 1920.

In the course of Kitabayashi's study of the choroid plexus in schizophrenia, he discovered an interesting heterotopia of the cerebellum and of the choroid plexuses, which up to the present time had not been described. Heterotopias and heterotaxias of the cerebellum are not rare. The one which the author describes differs in some respects from those heretofore reported. The patient from whom this particular specimen was obtained was a male schizophrenic (catatonic), 29 years of age, who died of tuberculosis after an illness of five years.

Three distinct heterotopias were noted. The first of these was located in the region of the tuberculum acusticum, the condition being seen best in the section cutting this structure, the corpus restiforme and the velum medullare inferius. The wedge-shaped projection of the flocculus contained heterotopic portions of choroid plexus embedded partly in the granular layer and partly in the medullary substance. No ependymal cells were included with the heterotopic villi.

The second anomaly was located in the oral portion of the horn of Ammon near the fimbria, and consisted of two abnormal cavities, in the form of a figure eight, filled by irregular masses of plexus villi. These spaces also were not lined with ependymal cells.

The third was located in the medullary substance, midway between the corpus geniculatum externum and the nucleus caudatus, about the location of the taenia semicircularis, where a small cavity was seen, which was connected with the ventricle and into which the villi of the choroid plexus projected filling it entirely. This cavity was lined with ependymal cells. Microscopic examination showed the heterotopic villi to be practically normal in structure. The third heterotopia, it was believed, might have arisen in a late embryonic stage or might have been acquired even later; the first two, however, must have developed at an early embryonic period on the basis of misplaced cells of the choroid plexus.

WOLTMAN, Rochester, Minn.

HEMORRHAGE INTO THE SPINAL CORD AT BIRTH. CHARLES W. BURR, Am. J. Dis. Child. 19:6 (June) 1920.

The first case reported is that of a boy, 4½ months old, who had four convulsions in the twelve hours prior to admission to the hospital. The previous history disclosed the fact that the birth had been a breech presentation, and the right shoulder had been hurt in delivery. The symptoms had apparently been present since birth, but had been overlooked. Examination demonstrated the position to be typical of that of a transverse cervical cord lesion; there was palsy of the external muscles of respiration with flaccid paralysis of both legs, with reflexes of defense, and anesthesia of the legs and trunk. A roentgenogram of the spine was negative. Lumbar puncture produced a fluid at first clear but tinged with blood at the end, containing ten cells. The Wassermann test was negative. The child died of pneumonia. At necropsy no signs of vertebral injury nor signs of old extradural hemorrhage were found. The cord and membranes from the fourth cervical to the first dorsal were a soft fibrous band. No hemorrhages were found in the viscera. Microscopic examination by Dr. A. J. Smith demonstrated on section of the involved area

that gray matter was not recognizable. There were no multipolar cells and only a few patches of degenerated white matter with no normal white matter so that at this level the cord was practically destroyed. There was ascending and descending degeneration above and below the site of the lesion.

The second case is similar as to history and physical findings, but no necropsy was obtained.

The author concludes from the necropsy findings of the first case that there was a hemorrhage into the cord and a subsequent myelitis, and he is inclined to the belief that the hemorrhage was spontaneous and was not caused by direct violence but by rupture of overfull vessels from pressure on the soft parts during birth.

WINKELMAN, Philadelphia.

UN CAS DE PARALYSIE SYPHILITIQUE DU NERF CUBITAL A LA PERIODE TERTIAIRE (SYPHILITIC PARALYSIS OF THE ULNAR NERVE DURING THE TERTIARY PERIOD). PIERRE ROBLIN, *Bull. méd.* **34**:838 (Sept. 18) 1920.

The writer has found only four records of syphilitic paralysis of the ulnar nerve in the literature. Gaucher has described the condition and considers it a secondary stage manifestation. In the case which Roblin reports, the condition was clearly a tertiary stage development.

A man, aged 38, without any previous knowledge of syphilitic infection, was seen by the writer in 1909, because of a slowly enlarging, painless, hard tumefaction of the lower lip. It was pronounced syphilitic, and under mercurial treatment disappeared in about forty days. Treatment with considerable vigor was then continued for two years.

Approximately six months later, there developed a right ulnar neuritis with paralysis. The features, typical of an ulnar (incomplete) lesion, need not be enumerated. They included pain on pressure over the hypothenar eminence. It was established that the lesion was not due to a bony exostosis which had "caught" the nerve. There was a dilatation of the left pupil which, however, reacted well to light. The treatment included potassium iodid and injections of mercuric benzoate in two series; and locally, massage and electricity. This led to a rapid amelioration, and the affected movements became almost normal in two months and completely so after five months. At the time, the inequality of the pupils remained.

The writer asks whether the pupillary change compels one to diagnose a central nervous system lesion, or whether the ulnar lesion was actually peripheral as appeared to be the case.

DAVIS, New York.

THE GERMAN INSTITUTE OF PSYCHIATRIC RESEARCH. EMIL KRAEPELIN, *J. Nerv. & Ment. Dis.* **51**:6 (June) 1920.

Kraepelin, in this article, stresses the need in Germany of extension of the scope of work done at the Institute for Psychiatric Research at Munich. Inquiry is needed into the many prenatal causes of disease with particular emphasis on the rôles played by alcohol, syphilis, infections and injuries, as well as the inheritable deficiencies which have heretofore received little attention. There is a broad field for investigation in the line of preventive medicine and biochemistry. It is stated that the investigations must necessarily reach out into every phase of the physical and mental life of the people: the

economic, developmental, religious, educational, artistic, literary and national life. Incidence of birth rate and control of death rate, marriage, and age of mortality should be sought out through statistical research in order that there might be constructed "a general picture of the folk soul." The research should aim to "make clear the nature and the sources of mental disturbances" and then "discover ways of preventing them, healing them, or making them easier to bear."

The furtherance of such work necessitates adequate appropriation for the endowment of positions and the work must be so arranged that younger men would be attracted to the field, who would be able to give their entire time to it, free from all other conditions which might interfere, such as earning a livelihood by other means. To the existing departments in serology, anatomy, and demographic-genealogical research, it is proposed to add departments in chemistry and psychology.

The author strikes the keynote in psychiatric research when he emphasizes the demands for inquiry into the underlying factors and predisposing causes of disease. Preventive medicine, although well recognized, has many limitations thus far, and greater cooperation and added facilities are requisite to make it more efficacious. This condition can be brought about through appropriations from the state and the enactment of necessary laws to enforce preventive measures. The outline of the work and suggestions given in this paper undoubtedly echo the thoughts and desires of many American psychiatrists, who are still struggling to place research in mental disease problems on a better scientific basis.

PATTEN, Philadelphia.

ANGINE DE POITRINE GUERIE PAR LA RESECTION DU SYMPATHIQUE CERVICO-THORACIQUE (ANGINA PECTORIS CURED BY RESECTION OF THE CERVICAL SYMPATHETIC NERVE).
T. JENNESCO, Bull. de l'Acad. de méd. **84**:93 (Oct. 5) 1920.

The symptoms of angina are caused by irritation of the cardio-aortic plexus by a lesion in the aortic wall. The reflex which arises from this lesion brings about vascular, painful and motor disturbances which constitute the syndrome known as angina. These disturbances can occur only when the nervous reflex originating in the aorta can reach mesencephalo-medullary centers.

By destroying the centripetal pathway for this reflex through the resection of the cervical sympathetic nerve (left), the anginal attacks are no longer possible.

Full data is given of a man aged 32, a syphilitic, with a specific aortitis and a history of severe frequent anginal symptoms, not benefited by mercurial treatment. The removal of the left cervical sympathetic nerve relieved him abruptly of these attacks. He was seen four years after the operation, and relief had continued during that period.

The writer believes that sudden death, so frequent in angina, is due to bulbar anemia provoked by spasm of the arteries; this is in turn a reflex from aortic irritation. In that event resection of the cervical sympathetic nerve in these cases would appear to be valuable not alone to relieve anginal pains, but also to prevent the occurrence of sudden death.

This therapeutic procedure for angina has never before been carried out.

DAVIS, New York.

TRAITEMENT DU MYXOEDEME (TREATMENT OF MYXEDEMA).
SAINTON, *Progrès méd.* **35**:409 (Sept. 18) 1920.

The only practical mode of administration of thyroid substance today for the amelioration of myxedema is the ingestion of the dried extract. The introduction of glycerin extracts or of thyroid lipoids is dangerous. The administration needs to be discontinuous and varied. During an initial period of adaptation careful recording of the pulse rate, etc., establishes a dosage. During a second period, the patient takes the fixed dose uniformly except for an interruption of a week in each month. During a third period, the dose is diminished and the intervals spaced.

In an adult, when the initial cause of the myxedema is an infection, the dosage of extract will be from 10 to 50 cg. In the myxedema of the infant (3 or 4 years) the dose is 2 to 5 cg.

At the time of puberty, thyroid opotherapy finds aid in the associated use of other endocrinal extracts. Especially in children, calcium, iron, phosphorous or arsenic preparations are useful adjuvants.

Attention is called to the occurrence of a syphilitic myxedema, and in this specific treatment is recommended.

The efficacy of opotherapeutic treatment of myxedema increases in direct ratio with the age at onset.

DAVIS, New York.

ON THE GROWTH OF THE NEURONS COMPOSING THE GASSERIAN GANGLION OF THE ALBINO RAT, BETWEEN BIRTH AND MATURITY. KENJI NITTONO, *J. Comp. Neurol.* **32**:231 (Oct. 15) 1920.

This paper is a continuation of the studies of the growth of the neurons forming the cranial and spinal ganglions of the rat now in process at the Wistar Institute. The author summarizes his conclusions as follows: The largest cells of the gasserian ganglion show three phases in growth: (1) rapid growth from birth to 20 days, (2) slower growth from 20 to 80 or 100 days, (3) a final phase in which growth is slow, or even a slight atrophy may occur. Growth of the nucleus is slight. Morphologic maturity of the cytoplasm is attained at about 20 days, but the nucleus-plasma ratio, which is high, more than doubles between birth and puberty. Before 80 days the volume of the ganglion cells increases as the area of the head increases. The diameters of the nerve fibers in the root of the fifth nerve are greater than in any of the branches. The fibers grow after the cells have stopped growing. The neurons in the gasserian ganglion differ in various size relations and in the period of growth from those in the ganglion of the seventh cervical nerve. They mature earlier.

HERRICK, Chicago.

PARALYSIS OF SIXTH NERVE WITH OTITIS MEDIA. WILLIAM ZENTMAYER, *Am. J. Ophth.* **3**:766 (Oct.) 1920

Dr. William Zentmayer presented the following case of Gradenigo's syndrome: C. S., aged 32 years, a milk dealer, had an attack of otitis media beginning Jan. 3, 1920. When seen by George M. Marshall, in consultation with his family physician, Dr. C. B. Schoales, on January 19, there was a copious thick purulent discharge and the upper and posterior wall of the canal was bulging. A simple mastoid operation was done on January 23. The night following the operation the patient had trouble with the left eye and

paralysis of the external rectus was noticed the next morning. On March 8 there was a complete paralysis of the external rectus. The author speaks of the syndrome and points out the situation when it is likely that the nerve is involved. He states that the paralysis may clear up in a few days or at once, following mastoid operation, but it usually persists for weeks or months.

REESE, Philadelphia.

THE INTERMUSCULAR NERVE CELLS OF THE EARTHWORM.

A. B. DAWSON, *J. Comp. Neurol.* **32**:155 (Oct. 15) 1920.

Since the earthworm is so often used as a subject for the study of fundamental physiologic problems and might to advantage more often be so employed, the details of its nervous organization are of general interest. Dawson describes four types of nerve cells of the peripheral nervous plexus in the muscular layers of the body, in addition to the sensory cells of the epidermis. One of these types resembles the latter elements, and these are interpreted as deep-lying sensory cells. The other three types are regarded as motor cells. This arrangement would provide for local peripheral reflexes without participation of the central nervous system, and the author considers the peripheral ganglionic nerve plexus as a survival of an ancestral diffuse nerve net similar to that found in the coelenterates. That is, when the central nervous system was formed by condensation of the primitive diffuse nerve net only a part of the primitive nervous system was so transformed, the remainder persisting as the apparatus here described.

HERRICK, Chicago.

DE LA PHYSIOLOGIE PATHOLOGIQUE ET DU TRAITEMENT DE LA MIGRAINE (PATHOLOGIC PHYSIOLOGY AND TREATMENT OF MIGRAINE). G. DIDSBURY, *Progrès méd.* **35**:429 (Oct. 2) 1920.

The writer discards toxic, general or even endocrinal theories as an explanation of migraine; he considers that it is caused by a local and superficial condition. In this he follows Nordström. The true cause of migraine must be sought in the presence of localized points of hyperesthesia, and he finds them along the course of the superficial nerves of the neck, cranium or face, and especially at the point of emergence of the nerves. In these locations lesions partaking of the nature of combined subcutaneous cellulitis, a myositis and an interstitial neuritis are present.

The author says that these foci can be cured by massage and believes that when they are disposed of, the migraine does not recur.

DAVIS, New York.

ACCESSORY SINUS DISEASE AND CHOKED DISK. HARVEY CUSHING, *J. A. M. A.* **75**:237 (July 24) 1920.

In view of the numerous unhappy consequences of indiscriminate and ill-advised operations on the accessory sinuses that have come to the author's attention, he feels obligated to express an opinion on the matter. Infections of the ethmoid cells set up definite types of optic neuritis, but when a choked disk (Stauungs-papilla; papilledema) is present, other factors than a simple inflammation are involved. Evidence of increased intracranial pressure caused either by an inflammatory process within the cranium or by a neoplasm should be considered. The lesion should be treated neurologically instead of by tampering operations on the nose.

ADSON, Rochester, Minn.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 21, 1920

EVERETT FLOOD, M.D., *President*

THE INFLUENCE OF THE CEREBRUM ON GROWTH. Presented by
DR. A. MYERSON.

This paper dealt with the infantile hemiplegias, the spastic hemiplegias occurring in infancy and in fetal life. It has long been known, though it has not appeared to any great extent in textbooks, that when the cerebrum is injured in fetal life by hemiplegia or inflammation, a limitation of the growth of the side of the body governed by that portion of the cerebrum takes place; the limbs tend to be shortened, the bones to be thinner and the face to be asymmetrical. The ear and the breast are not affected, although in certain cases they are smaller on the involved side. The diameter of the chest is less, and the scapula and clavicle are shorter. Roentgenograms show no particular pathologic phenomena, although they sometimes show that the ossification process seems to lag behind.

Freud's essay on this subject is classic. It is interesting that Freud's first researches should have been in the domain of physical injuries, while his later contributions were on the psychic injuries in fetal life. Perhaps there is a connection beneath the surface.

The phenomena to which this paper calls attention, are first, the contrasting conditions of the hand in adult and infantile hemiplegia. In adult hemiplegia a marked spasticity in the fingers usually develops—the typical hemiplegic hand. In infantile hemiplegia there is hypotonia, which is most marked and constant. This seems worthy of notice first, because the rule in infantile hemiplegias is contrary to the rule in the adult, and second, because it is a constant phenomenon and practically never absent in some degree or other. It is therefore one of the ways of differentiating between infantile and adult hemiplegia.

Another phenomenon concerns the scapula. William Graves of St. Louis has made his life work the study of the condition known as the scaphoid scapula, in which the ordinary convex vertebral border of the scapula is concave in construction, the angle of the spine is changed and the bone itself is smaller and thinner. In his early papers he laid some stress on syphilis as a cause, but now he considers the condition a phenomenon of general pathology. Critics of his work have stated that the shape of the scapula is dependent on use and that the concavity is brought about largely through use. The series of cases which were studied at Dr. Fernald's Institution, Massachusetts School for the Feeble-minded, showed that the arm and leg may be completely paralyzed and that there may be no function on that side. The scapulae are also shorter and thinner. They are, however, of the same shape on the affected and the unaffected sides. If the scapula is scaphoid on the sound side, it is the same on the paralyzed side; if the scapula is "normal" on the sound side, it is the same on the affected side. In other words, the shape of the scapula does not depend on use, which is Graves' contention.

OPPORTUNITIES FOR CREATIVE EFFORT BY THE MASSACHUSETTS SOCIETY FOR MENTAL HYGIENE. Presented by DR. A. W. STEARNS.

The Society for Mental Hygiene in this state was one of the first to be organized. It has been an important factor in the development of the National Committee for Mental Hygiene through its membership and its opportunity to help because Massachusetts is so far advanced in these matters. In fact the question has been raised as to whether there is a place in a state so highly organized as Massachusetts for a private society for mental hygiene. This question can be answered in the writer's opinion in the affirmative.

There are many fields of activity which a private society can enter quite apart from those covered by a state organization. However, the excellent development of our state organization makes the function of the Massachusetts Society for Mental Hygiene quite different from that of those in some other states.

The aim of future efforts should be: (1) To create and maintain an enlightened public opinion concerning the relations of mental normality and abnormality to a useful life in the community. Mental disease and psychology are still surrounded with mystery in the minds of the general public. One of the greatest handicaps of mental disease is the attitude which even the most enlightened people take toward it. Efforts at propaganda by state departments often arouse suspicion on the part of the public. There have been occasions in this state during the last few years when resentment has been created by proper attempts of state organizations to stir up public opinion. A private society organized primarily for educational purposes does not meet this opposition. There is little occasion in this state to conduct a campaign concerned primarily with institutional care of the insane, but public opinion is still archaic in its attitude toward the insane or the defective person in the community. It is surprising to find the number of intelligent people who think that many of the insane are kept in institutions by their relatives in order to get their money. The attitude of the public toward an insanity plea in court is hardly intelligent. Again, a failure to recognize the need of adjustment in society for the aged and psychoneurotic leads to broken home ties, divorces and endless litigation. There is evidence to show that the suicide rate can be influenced by propaganda.

(2) To maintain the highest standard in our state institutions. This society should be in an independent position enabling it to commend or criticize when such action is for the best.

(3) To foster research and investigation tending to increase the knowledge of this subject and in that way ultimately to reduce burdens. Every man in this state who develops a new or promising idea, if practical, should feel that this society stands ready to lend its assistance. The routine duties of the state service and the tremendous problem of feeding and housing so many dependents occupy the major part of the time of those officially dealing with the insane. Thus, a society of this sort may well conduct or patronize research.

(4) To extend the investigations now being made in a few centers on the relation between mental disease, personality and crime. There is a tremendous field for mental hygiene in the administration of justice and the practice of law. The good beginnings in this field should be supported and new ones developed.

(5) To take an advanced stand concerning the prevention of feeble-mindedness. The general question as to segregation, sterilization and control of reproduction of the feeble-minded can perhaps best be answered and leadership assumed by a private society.

(6) To extend special classes now so successful in a few places. Laws have been passed and state bureaus are now engaged in work with the defective and backward in our schools. The public must be prepared for this innovation by being informed and encouraged to use special classes properly.

(7) To formulate and to carry out organized effort for the care of the handicapped in the community through social service. The last fifty years have been characterized by a tremendous institutional development. The public is now willing to trust the handicapped one to the care of an institution, but further extension is almost prohibited by the question of expense. There are signs everywhere that the contribution for the next generation may be for economical reasons the perfection of community care of the handicapped. Experience has shown that private agencies dealing with a few cases in a personal way can often develop a method more readily and carry on experiments with less risk than can the more cumbersome machinery of a governmental department.

(8) To promote the establishment of courses on mental hygiene in the professional and normal schools. Courses in hygiene are given in many colleges in Massachusetts. These courses have largely to do with the body and should include the mind. Physicians, lawyers and school teachers are continually going forth to their work with too little equipment along these lines. The "urge" to greater effort may well come from our society.

(9) To emphasize the need of mental hygiene in the industries. Individuals succeed or fail according to their mental capacity and ability to adapt themselves to their work. Business houses and industries are employing almost any sort of a person who thinks he can help. This field should be gone over, studied and the fundamentals made matters of routine.

Lastly, there are in this state a great many persons who are professionally or otherwise engaged in work related to mental hygiene. These should be organized, kept informed and asked to advise and so become a greater factor in the progress of our state. Among them are included clergymen, lawyers, doctors, philanthropists, teachers, employment managers, social workers and innumerable others who may be given an opportunity for self-expression through membership in a society for mental hygiene.

IMMIGRATION FROM A MENTAL-HYGIENIC STANDPOINT. Presented by DR. A. J. NUTE.

The earliest migratory movements were due to a class of people known as colonists, as well as immigrants, who sought economic benefit and religious freedom. The character of the migratory movement of a later day has changed so that instead of seeking political rights or religious freedom many are ignorant of the simplest facts regarding the government under which they formerly lived and are apt to confuse the American flag with the dollar bill. The colonist may be classed as an immigrant who was willing to endure the hardships of climate, famine, disease and hostile Indians in order to gain a home and his freedom. The immigrant may do this also, but he is more likely to profit by what the colonist has started. Therefore the present day tendency is to settle in the cities where it is usually possible to live under

conditions more or less satisfactory. In the early settlements selection was the rule; kindred spirits flocked together. Later, as the country grew, the selective element changed and a restrictive policy had to be enforced. The earliest laws passed by the United States, from 1819 to 1882, were largely to protect the immigrant. Since that time laws have been passed largely to protect the United States. In the early days each state had to protect itself as best it could. This was unsatisfactory without national action because only Congress could regulate commerce with foreign countries. New York took the lead and finally Congress was induced to take such steps as would start an organized, uniform immigration policy. At first this was carried out by the states by agreement with the government. In 1890, the federal government assumed control and from that time the laws have gradually tended to exclude the more undesirable types and to protect the desirable.

Traveling in the early days of the sailing ship was a hardship but little appreciated today. The immigrant furnished his own food, and a death rate of 10 per cent. during the voyage was considered normal. From time to time the various countries improved these conditions so that before the days of steamships it was required that immigrants should be provided with certain space, certain food, and some protection in regard to sanitary matters and the competency of ship's officers. Since the invention of steamships the general sanitary and living conditions have improved until at present the so-called steerage passenger travels better than the cabin passenger of one hundred years ago. On arrival at the immigration station means are taken to protect his rights and interests.

In regard to the laws relating to mental inspection, mental defectives have been mandatorily excluded for a comparatively short time. The feeble-minded might enter legally up to 1907. Under the laws of 1882 idiots and the insane were the principal mental defectives excluded. In 1893, the law was made more rigid relative to information in regard to arriving aliens. In 1903, epileptics and persons who had been insane within five years and persons who had had two or more attacks of insanity were also barred. In 1907, in addition to the foregoing, a person having a mental defect that might affect his ability to earn a living was excluded, and for the first time a penalty was placed on the steamship company bringing to this country such mental defectives as idiots, imbeciles and epileptics. Provision was also made to deport, at any time within three years after date of entry, any alien that entered the United States in violation of the law or who became a public charge from causes existing prior to landing. The last act of 1917 added constitutional psychopathic invalidity and chronic alcoholism, those having had a previous attack of insanity at any time and persons not coming under the above departments who could be classed as mental defectives. The alien was given the right to appeal when certified to be mentally defective and allowed to present one medical expert in his behalf before a medical board. The penalty or fine for the transportation company was increased, and in addition the deported alien was entitled to a refund from the transportation company of a sum of money equal to that paid by him for transportation from the port of departure to the port of arrival. The provision made to deport any alien was extended from three to five years and the wording changed from "causes existing prior to landing" to "causes existing not affirmatively shown by himself or friends to have arisen subsequent to landing."

The inspection may be likened to a sieve rather than a dam. The immigrant has had at least one and maybe several examinations prior to embarkation. A well-trained examiner can tell at a glance the nationality or race of

the persons who appear before him. This is necessary in order that he may know or recognize the normal from the abnormal. Those detailed are examined in a quiet room after twenty-four hours of rest. Physical examinations are made to make allowance for physical defects. A brief mental examination is given to ascertain the amount of acquired knowledge and to test the mental activities. By this method the normal are released with as little delay as possible. Examinations are then made by at least two doctors on different days, and a certificate is issued when all agree as to the findings. As long as doubt exists reexaminations are made.

DISCUSSION

DR. DONALD GREGG remarked that on shipboard it was extremely interesting to watch immigration inspectors quickly go over two or three hundred people and by simple observation pick out physical and mental defects. He asked Dr. Nute to mention more specifically the general signs that an inspector would look for in examining several hundred men.

DR. A. W. STEARNS objected to the inference that we were all immigrants. The publicity that has been given during the last year to the early history of this country would tend to show that this is not so. Several persons have attempted (Dr. James J. Putnam in particular) to define or describe the New England character. The population of New England was not an immigrant population but grew through reproduction. For a period of twenty years about 20,000 people came to this country and then, with the exception of the Scotch Irish, immigration stopped. As many returned to Europe as came so that for nearly 200 years the increase of population was by reproduction. This was also accompanied by extreme isolation. There were no large cities, and the settlements of colonists were widely separated so that a type grew up which has become quite distinct from the immigrant type.

Dr. Stearns also suggested that great differences of mentality in children and illiterates could be detected by skilled observation of the facial expression and manner. He has found this to be true in his experience with the lowest type of Southern negro.

DR. A. J. NUTE, in answer to Dr. Stearns, said that it was true that facial expression was a guide to intelligence. For instance, if an intelligent immigrant child of about 2 years is brought into a room hung with bright flags his passive expression at once will change to an animated one. A simple toy will give the same result.

In reply to Dr. Gregg's question, Dr. Nute said that an experienced inspector must be familiar with the types of people he examines. He must be able to tell at a glance the nationality of the man passing before him; if he cannot do this, he is not a competent examiner. The stolidity which is to be expected in a Pole, for example, might be indicative of a dementia in an Italian. If an Italian woman had the flush of a Scandinavian, she would be taken aside in order to discover whether she had a temperature. Again, the immigrant dresses in his best when he comes into port. He wants to make the best showing he can. If one comes along carelessly dressed and appearing indifferent, it immediately attracts attention and it is well to investigate the mental attitude of that person. Now the gross defects are fairly well eliminated on the ship, and there is time to devote to the finer defects. This fact is responsible for the large increase in the detection of the mentally defective.

PHILADELPHIA NEUROLOGICAL SOCIETY

*Regular Meeting, Oct. 22, 1920*SAMUEL D. INGHAM, M.D., *President*

A CASE OF EPIDEMIC ENCEPHALITIS OF THE PARALYSIS AGITANS TYPE. Presented by DR. F. H. LEAVITT.

Dr. Leavitt said that the case was of interest because the spinal fluid examination showed a positive Wassermann reaction, a cell count of 22, a positive globulin reaction, and a colloidal gold curve of 3455432110, while the blood Wassermann reaction was negative.

The previous history was negative. The patient was 31 years old and married. Feb. 21, 1920, his temperature was 103 F., which was followed by a general "neuritis," with severe pains in all parts of the body and some delirium for two weeks. He had diplopia for weeks. He had incontinence of urine and feces for seven weeks. A trophic bed sore developed after five weeks, which did not heal. Gradually a deformity of the feet developed, which prevented the patient from walking. The pain disappeared entirely in three weeks and was followed by tremor of the hands and monotonous speech. A little later tremor was noticeable in the legs, and a paralysis agitans picture developed and has persisted ever since.

There was constant tremor of the toes of the left foot and of both arms, more marked on the left and accentuated by emotion and voluntary movements. The facies were mask-like and expressionless, and the speech was monotonous and tremulous. He could move all muscles of the face voluntarily but to a limited degree. There was no difficulty in deglutition or phonation. All movements of the extremities were weak and slowly and awkwardly performed. Both feet were held in the position of talipes equino-varus. Stroking the sole of the foot caused withdrawal of the entire leg but no movement of the toes. There was no ankle clonus. The knee jerks were equal but weak. Cremasteric and abdominal reflexes were normal. Sensibility to touch, heat, cold and pain was normal. There was a large sloughing bed sore over the sacrum. The sphincters were controlled. The results of ocular examination were negative. Hearing, vision and taste were normal.

During his stay in the hospital, otitis media with peripheral cellulitis, caused by a streptococcus pyogenes infection, developed, which subsided two days after the drum was incised.

After treatment by mercurial inunctions and potassium iodid in small doses, the blood and spinal fluid Wassermann reactions were negative; cell count, 2; globulin +; colloidal gold 111112100.

In the majority of cases of epidemic encephalitis the spinal fluid reaction has been negative, but in this patient it was positive, and it has not been proved that the encephalitis could not cause the reaction. Furthermore, the patient did not improve under antisyphilitic treatment, but rapidly and progressively became worse. Neurosyphilis does not produce a clinical picture simulating paralysis agitans as did the picture in this case.

DISCUSSION

DR. CHARLES W. BURR said that the name lethargic encephalitis was not used because he believed that it was misleading since many of the patients never had lethargy. Epidemic encephalitis is a better designation. Instead

of lethargy, the patient may be delirious or somnolent during the day and delirious at night. When Dr. Burr first saw this man in bed and saw only his face, he thought it was a case of paralysis agitans. At that time Dr. Burr knew nothing about his history. Then the patient had the same tremor in his hand that he now has in his foot. It was not surprising that the spinal fluid Wassermann reaction was negative under treatment. Dr. Burr wondered whether encephalitis could make a latent syphilis active, just as in childbirth a latent malaria in the mother may be made active, or whether in encephalitis apart from syphilis there may be a positive Wassermann reaction due to the encephalitic infection itself.

DR. JOHN H. W. RHEIN said that the spinal fluid examination had been made in a large number of cases reported in the literature, and a negative Wassermann reaction had been found in all instances. In one case which he reported, there was a positive spinal fluid Wassermann reaction but the patient died and no necropsy examination was made. He regarded the rare occurrence of a positive Wassermann reaction in epidemic encephalitis in the spinal fluid as a coincidence.

TWO BOYS, BROTHERS, WITH CHOREIC ATAXIC MOVEMENTS.

Presented by DR. J. HENDRIE LLOYD.

Dr. Lloyd said these cases might perhaps be instances of atypical Friedreich's ataxia, although he was not sure that this term was the proper designation for them. The father and mother were well and had had eight children, of whom the other six were well. The patients did not come next to each other in order of birth, there having been two healthy children between them. There was no family history of any such disease on either side.

The condition of the elder brother, 20 years old, began with irregular movements when he was 4 years of age. These had greatly increased during the last three years, so that he was almost unable to walk. His gait was rather reeling or staggering, not unlike a cerebellar gait. He had free, even increased, knee jerks, but the ankle reflexes were diminished and there was no Babinski reflex. There was vertical nystagmus; the eyegrounds were normal. The pupils reacted to light and on accommodation. There was a speech defect, not typical of Friedreich's ataxia, and the boy was rather dull in mind. There was no pain or sensory disturbance. As he sat there was a slight scoliosis. The Bárány test (made by Dr. Hunter) showed that the impulses went through all pathways, but after rotation the nystagmus was of unusual duration (48 to 57 seconds). Past pointing, falling and hearing were normal. The Wassermann tests of the blood and spinal fluid were negative.

The younger brother, 14 years old, had movements that were rather more choreiform than those of his brother. They involved the face as well as the limbs, and dated from an early age, not accurately stated. He was able to walk without difficulty. He also had a slight speech defect, little, if any, nystagmus, and the pupils reacted normally. The Bárány tests showed rather prolonged nystagmus after rotation. There was some past pointing. The knee reflexes were present; the right was decreased.

The question is whether there is a cerebellar lesion in these cases, or whether they are of a purely spinal type. If the former, they would probably approach Marie's cerebellar ataxia. The preserved, even increased, knee jerks rather point against Friedreich's ataxia, although some such cases have been reported.

DISCUSSION

DR. F. X. DERCUM said he had shown a patient before the Neurological Society about three years ago, a man of 28, whose symptoms, beginning at 6 years of age, were very much like those of the elder of the two boys shown by Dr. Lloyd. Dr. Dercum had regarded the case as in all probability one of hereditary cerebellar ataxia. There was a marked family history. Dr. Dercum thought it quite likely that hereditary cerebellar ataxia and Friedreich's ataxia were closely related conditions, as in both it would seem that there are feebleness of development and consequent degenerations, essentially biologic in nature. (Dr. Dercum's case was published in the *New York Medical Journal*, Aug. 25, 1917.)

DR. JOHN H. W. RHEIN said that some years ago he had had an opportunity to study a family group with spastic paralysis. At that time he went over the entire literature of family groups with conditions of the spinal type. It seemed to him at that time that there was great confusion in the classification of these diseases and it appeared possible to group these cases so that at one end could be placed typical Friedreich's ataxia and at the other, typical spastic paraplegias with several different types, more or less sharply defined, in between. Between these two typical groups, there were cases in which the arms were involved; others in which the cerebellar symptoms were added; still others exhibiting bulbar symptoms; another group showed muscular atrophy; another group had symptoms that resembled those of disseminated sclerosis. It seemed to him that these groups were mere subdivisions of the same disease, and all represented probably the same pathologic process.

DR. WILLIAM G. SPILLER said that while these cases presented some features atypical of Friedreich's ataxia, he thought it a mistake to discard, or to put in a class apart, certain cases of this disease in which a few uncommon symptoms occurred. Friedreich's ataxia is capable of many variations; it is a posterolateral sclerosis, possibly the best example of a true systemic combined sclerosis, and the symptoms vary as the lesions predominate in one system of fibers or another. The degree of implication of the cerebellum in this disease has been a matter of dispute during many years. While it is desirable to recognize the distinctions between similar diseases of the nervous system, it is equally important to recognize the connecting links.

DR. T. H. WEISENBURG said that Dr. Lloyd had given him the opportunity to study his cases before their presentation, and he believed that they were distinctly cerebellar and that the pathology consisted either in a congenital lack of development or atrophy in a definite part of the cerebellar system. The question had come up as to whether these cases could not be diagnosed either as Friedreich's ataxia or whether they belonged to Marie's hereditary cerebellar ataxia. Regarding Marie's type, he called attention to the fact that when Marie first described this disease in 1893, he based it entirely on the observations of others.

Analysis of the clinical records of these cases, as pointed out by Gordon Holmes, shows that they were by no means identical or even similar, and pathologic studies showed that in only one case, that of Fraser, was there evidence of acquired cerebellar disease. Dr. Weisenburg agreed with Holmes that no form of disease existed to which the term hereditary cerebellar ataxia could be aptly applied, and said it was apparent that this title has been a convenient pigeon-hole in which every obscure or even plain type of cerebellar disease has been placed.

There will never be progress in cerebellar diagnosis so long as neurologists persist in using the old time classification and symptomatology. Most modern reports of cerebellar cases are incomplete, and no attempt has been made to delimit cerebellar asynergy to parts of the body. The description of most cerebellar cases gives one the idea that asynergy and adiokocinesis are merely symptoms, whereas every one should know by this time that the primary attribute of the cerebellum is to synergize all motor functions and whatever symptoms occur are the result of loss of synergic action of certain muscles concerned in a definite movement.

DR. J. HENDRIE LLOYD said that the discussion had brought out just what he wanted. He agreed that the term Friedreich's ataxia admits of some variety. In Griffith's paper, written about thirty years ago, including most of the cases reported up to that time, a few were reported with exaggerated knee jerks; but not all the cases in that paper had been reported by competent observers. In Friedreich's ataxia a Babinski reflex had sometimes been observed; he had seen one such case. Marie's cerebellar ataxia had always seemed to him to be a rather different disease, nevertheless it could not be ignored in considering the present cases. The elder boy especially suggested the possibility of a cerebellar lesion. He agreed with Dr. Weisenburg that this whole subject of the familial ataxias needs careful revision. The members of the society would probably recall that less than a year ago Dr. Lloyd had shown two colored boys, brothers, with Friedreich's ataxia. One of these boys had since died of pneumonia, and sections from the cerebellum and cord will soon be ready for examination.

PROVOCATIVE SPINAL FLUID CHANGES IN NEUROSYPHILIS.

Presented by DRs. H. C. SOLOMON, Boston, and J. V. KLAUDER.

The provocative arsphenamin reaction in the blood serum is a familiar phenomenon. In many clinics when a patient suspected of having syphilis gives a negative Wassermann reaction on the blood serum, an injection of arsphenamin is given, and then the blood is tested at daily intervals thereafter, because it apparently has been shown in many cases of this sort that after the injection of arsphenamin the Wassermann reaction will be positive.

The purpose of this paper was to call attention to a similar reaction taking place in the spinal fluid. Case histories were presented in which after either intravenous or intraspinal injections, a negative spinal fluid became positive, or the positive pathologic findings were intensified by such treatment. From the clinical standpoint, intensification of symptoms relating to the central nervous system has been frequently described as occurring after treatment. To this phenomenon the term neurorecidive or neurorecurrence has been applied. One is not sure in these cases that the resulting intensification of symptoms is due to the introduction of the drug, or that it is a mere coincidence that an increase of symptoms appears following the injection. However, in secondary syphilis the intensification of symptoms following arsphenamin injection has occurred so frequently that it has been considered as due to liberation of toxins resulting from the lytic action of arsphenamin on spirochetes. This is the so-called Herxheimer reaction. If this can occur with skin lesions, there is every reason to suppose that the same might occur in neurosyphilis. However this may be, the following cases would seem to show that following intravenous and intraspinal injections of arsphenamin or arsphenamized serum, the spinal fluid findings may become more strongly positive.

These cases illustrate the fact that after antisyphilitic treatment a negative spinal fluid may become positive, or one that is slightly positive may give more strongly positive reactions.

In Case 1 the diagnosis was cleared by the effect of a provocative injection of arsphenamin. In this case the spinal fluid, which was negative prior to antisyphilitic treatment, became strongly positive in all tests after one injection of arsphenamin. It is worth while to note that the blood did not become positive. This result is also found in the other cases of this series.

Case 2 of the series shows the intensification of the spinal fluid Wassermann reaction after intravenous injection and also the provocation of a positive colloidal gold reaction, globulin and pleocytosis. It is perhaps of interest to point out in this discussion the curious fact that no provocative change was noted in the serum Wassermann reaction. In the light of present knowledge, perhaps, one can conclude that the patient is cured so far as visceral involvement is concerned. Latent foci of spirochetes in the nervous system apparently were activated by treatment, as was manifested by the provocative spinal fluid changes.

The patient in Case 3 presented so definite a picture of tabes that no doubt of the diagnosis could exist, despite a spinal fluid that was negative in all phases, with the exception of a small amount of globulin and albumin increase. No provocative result occurred either on the blood or spinal fluid as the result of intravenous injection, but intraspinal subdural injections of arsphenaminized serum provoked positive Wassermann and gold reactions. It is worth while to note that under treatment these reactions were reduced to almost normal. From the clinical standpoint, improvement took place even during the period in which the tests became more strongly positive. The patient, who was in the paralytic state of tabes, being confined to bed, became able to walk without a cane, although quite ataxic.

Case 4 was that of a patient with undoubted tabetic disease, showing negative blood and spinal fluid. Under intraspinal injections the spinal fluid showed positive globulin, albumin, pleocytosis and gold reactions, but the blood and spinal fluid Wassermann reactions remained negative. Contrary to the experience in Case 3, no improvement in the patient resulted from this treatment.

Case 5 was that of another patient in whom the clinical evidence made a diagnosis of tabes certain, but whose spinal fluid Wassermann reaction was negative. The blood, however, was positive, and there were 139 cells per c.mm. in the fluid. After intravenous injections of arsphenamin, the Wassermann reaction in the blood serum became negative while the spinal fluid Wassermann reaction became positive. The cell count decreased, but the colloidal gold reaction became more strongly positive. From the clinical standpoint, the result of treatment was eminently satisfactory, as all the pain disappeared, the ataxia became markedly improved and the patient was able to return to work after having been incapacitated for some time.

Case 6 showed the provocative results in the ventricular fluid after intraventricular injections, similar to that observed in the spinal fluid of the preceding cases.

Altman and Dreyfus have shown that arsphenamin may have a provocative influence on the spinal fluid in cases of primary and secondary syphilis. These writers speak of this phenomenon as a provocative neurorecidive. A criticism of this conclusion might be offered, as it is quite possible that the condition had nothing to do with the introduction of arsphenamin, but might have

occurred had this not been done. As this occurred some time after the injection, and as there was only one injection, this case cannot be considered as strong evidence, but as illustrative of this type of reaction.

A study similar to that of Altman and Dreyfus was made by Dr. Klauder. In a series of twenty-five cases of secondary syphilis, the spinal fluid was examined before and after a course of four intravenous injections of arsphenamin, administered at intervals of from seven to ten days. Provocative spinal fluid changes were observed in some of the cases on the second examination of the spinal fluid. This observation and those of Altman and Dreyfus are in accord with the hypothesis of Gennerich that arsphenamin is capable of producing provocative spinal fluid changes in the early period of syphilis.

The change of a spinal fluid by arsphenamin therapy from negative to positive is, in all probability, the expression of a Herxheimer reaction. A neurorecidive or neurorecurrence following arsphenamin injection is probably a clinical expression of the same reaction. The apparent provocative reactions in the spinal fluid of neurosyphilitic patients, as herein reported, would seem to be of a similar nature and in line with the above. It should be emphasized that this provocative reaction is not a frequent occurrence, even in cases in which there is evidence of central nervous system involvement with negative findings in the spinal fluid.

Case histories of neurosyphilis were presented in which no provocative spinal change was noted following treatment with arsphenamin.

A note was made of cases of neurosyphilis which were made worse by arsphenamin therapy. These reactions doubtless manifest the powerful spirocheticidal action of the drug. The arsphenamin either activates foci of spirochetes or, as a result of the lysis of great masses of organisms, the toxins are liberated in large amounts, which produces pathologic results. We may, then, assume that this result is expressed in a laboratory way by the appearance of positive reactions in the blood and spinal fluid, and in a clinical way by the appearance of neurorecidives and the accentuation of symptoms.

DISCUSSION

DR. H. C. SOLOMON, Boston, said that he would like to say a word in relation to the effect of arsenic in the production of neurorecidives. For a time there was in Boston general acceptance of the idea that arsphenamin produced deafness and blindness. Dr. Crockett of the Massachusetts Charity Eye and Ear Infirmary made a study of syphilitic nerve deafness before the arsphenamin era and after. He found the incidence was essentially the same after the introduction of arsphenamin, if anything a little less, and Dr. Solomon agreed with this conclusion. For the past year and a half he had in a sense dropped the rôle of neurologist and had become a syphilologist in order to learn what he could about these problems.

In the Massachusetts General Hospital it was the rule to put every syphilitic patient on arsphenamin at once. This treatment was followed up by mercury. They did not find that many of the patients developed nervous system lesions after treatment was begun. Dr. Solomon said he had seen many patients with secondary syphilis, untreated, who had thrombosis of various cerebral vessels occurring within three to six months. He saw an occasional patient who had received treatment and developed cerebrospinal involvement during the secondary period.

It was obvious that half a dozen injections of arsphenamin would not help a paretic. Six years ago Dr. Solomon began to give from 0.6 to 0.9 gm. of

arsphenamin twice a week over a period of from six months to a year. He had patients now who began to receive treatment six years ago and, contrary to the usual run of parietic patients, they are now happily at work. It surprised him that they had even not had kidney involvement. In many ways arsphenamin is a dangerous drug and should be used with care by those who are inexperienced in its use. Medical examiners in Boston gave a warning, "You would better not treat patients at your office unless you know what you are doing." Sudden deaths have occurred from the use of arsphenamin. Of course the same thing is true in surgery.

DR. J. HENDRIE LLOYD said that this was a most important subject. In the last few years he had seen in his service in the Philadelphia General Hospital four cases of deafness in early syphilis following the use of this arsenical drug. In two of them there was associated paralysis of the seventh nerve. In all cases deafness had occurred within a year of the primary lesion. He had been on the Philadelphia General Hospital staff for a great many years, and he had never seen such conditions until the introduction of this arsenical preparation. He only stated this for what it was worth. He showed two of these cases before this Society a year or two ago. Dr. Lloyd said the question arises "Are we doing this with our drug, or are we not?" It is one of the worst complications that can come in syphilis. Involvement of the eighth nerves causes total bilateral deafness and is incurable.

DR. WEISENBURG said that there was being developed in Philadelphia, and perhaps all over the country, a group of chronic arsphenamin takers who went from one clinic to another for injections. For example, in the Polyclinic Hospital, which has one of the largest syphilitic clinics in the country, there were quite a number of tabetic patients who came weekly for the injections of arsphenamin. A number had had over a hundred injections. These patients have the idea that the only method of cure is to receive weekly arsphenamin injections, and if one clinic refuses to give it to them, they go to another, frequently not telling the truth about how many injections they have received. Dr. Weisenburg was impressed with the ease with which these patients took the injections. They came practically without any preparation; after receiving the injection they left promptly, and often went to work directly after, without difficulty.

A CASE OF BRAIN ABSCESS CAUSED BY A POCKET KNIFE INJURY OF THE SKULL. Presented by DR. H. F. DUNLAP.

History.—A colored man, aged 19, admitted to the Philadelphia General Hospital, Sept. 11, 1920, complained of severe headache, pain through his eyes and stiffness of the neck. His past history was negative except for an injury to the left side of the head from an alleged razor cut received in a fight two years before. There were no sequelae. He denied venereal disease but admitted excessive alcoholic indulgence. The present illness began three weeks ago with severe and continuous generalized headache, dull aching pain in the eyes and stiffness in the neck and shoulders beginning one week later. Since that time he had had from twelve to twenty-four hour intervals of inability to talk or remember things but he had never become unconscious. There had been no vomiting or convulsions. This condition obtained at the time of his admission.

Physical Examination.—The pupils were unequal, the right being larger than the left, but they reacted well. The left palpebral fissure was narrower. There

was paralysis of both external recti, more marked on the left. Convergence was normal. There was bilateral choked disk of 4 diopters, with intense engorgement of the veins, a few white spots on the retina and a few scattered hemorrhages. The left brow showed slight paresis. Speech was slow, stammering and indecisive. The neck was markedly rigid. The tendon reflexes were all present and active; superficial reflexes were normal. No Kernig's sign and no ataxia were present. Sensation could not be tested on account of the patient's mental condition. He was lethargic and somewhat stuporous, although he could be aroused. He showed restlessness and irritability but slept a great deal. The blood pressure was systolic, 120; diastolic, 70. Leukocytosis ranged from 9,800 to 10,800 with 78 per cent. of polymorphonuclears. The blood Wassermann test and blood culture were negative. The urine examination was negative. A daily spinal fluid examination was made. The spinal fluid was always clear until the last specimen. The cell count ranged from 730 to 840, with the exception of the last specimen, which was 2,000 plus. The cells were mostly lymphocytes—from 84 to 93 per cent., with the exception of the last specimen which showed a strongly polymorphonuclear leukocytosis. The spinal fluid was bacteriologically negative, with the exception of the last specimen which showed *Micrococcus tetragenus* and later a staphylococcus. The globulin tests were strongly positive, though the Wassermann and Fehling tests were negative. The temperature ranged from normal to 101, and the pulse from 50 to 60 until the last three days when it mounted as high as 100. The patient was unable to cooperate and too sick to permit a roentgen-ray examination of his head.

Progress: The headaches persisted, although they were relieved by lumbar puncture to a certain extent. At times he showed a rather profound stupor, but on the third and fourth days he was more alert and even said his head was better, although his neck and shoulders were more rigid. On the fifth day he became disoriented, and the following day he was restless and delirious. He died on the sixth day after admission.

Postmortem Findings: The point of a pen knife, 2 cm. long by 5 mm. wide, projected from the inner surface of the calvarium at the left parieto-occipital fissure, extending down through the dura and into the brain substance. Thick creamy pus exuded from this point. The dura was markedly thickened and adherent here, both to the brain and the calvarium. In the left parietal lobe, just above the sylvian fissure and bounded anteriorly by the postcentral gyrus, above by the great longitudinal fissure and posteriorly by the anterior portion of the occipital lobe, was an abscess 4 cm. in diameter, whose external wall was the thickened dura, which formed a pyogenic membrane. The surrounding cerebral tissue was soft, and there was no sharp demarcation between it and the healthy tissue. The lateral ventricles contained a small amount of pus which on culture proved sterile. There was no exudate over the general cortical surface. Cultures from the abscess revealed *Staphylococcus aureus* and *Micrococcus tetragenus*.

DISCUSSION

DR. CADWALADER said this case showed that an abscess may develop long after the injury to the brain has been received. In such cases lumbar puncture may be useful to determine whether or not a diffuse meningitis is developing. A leukocytosis of the spinal fluid would indicate such a complication; and if it is present, the case is not a favorable one for operation. Meningitis often develops in such cases by the extension of the infection from the site of injury to the lateral ventricle of the brain.

DR. T. H. WEISENBURG said that when the patient was admitted, he presented many of the symptoms of lethargic encephalitis, and this diagnosis was tentatively made because of the stupor, sixth nerve palsy and the limb symptoms.

The presence of the piece of pocket-knife, which was found on stripping the dura from the calvarium, was not suspected. It is extraordinary that this should have been within his skull for two years without causing any symptoms previous to two weeks of his entrance to the hospital.

EXTRADURAL ABSCESS OF THE MIDTHORACIC REGION OF THE SPINAL CANAL SECONDARY TO A BOIL IN THE NECK. Presented by DR. WILLIAM G. SPILLER and DR. V. W. M. WRIGHT.

J. H., a white man, 36 years of age, was admitted to the Philadelphia General Hospital, Sept. 29, 1920. He began to have pains in his back August 23, over what he termed the kidney region. Up to this time he had enjoyed good health. He had chills and fever, which did not last long. He denied having had any injury to the spine or hip except a blow on his hip during August. About July 15, 1920, he had had a large carbuncle on the left side of his neck in its upper part, also two over the sacrum, one on the right upper limb and two on the left upper limb. He had slight stiffness in his back, following the blow to the hip, and severe pain over the lumbar vertebrae. This pain, which developed with chills, was severe; his back was so stiff that he could bend forward only with difficulty. The pain radiated up and down the lumbar region and anteriorly through the abdomen, and as it subsided it left the affected parts "dead." The pains then began to spread down the lower limbs and through the chest and by September 4 they were very severe. He described the pains as "thunder and lightning"; they came like a flash and felt like "fire and needles." They ceased on the twenty-fifth day (September 22). Paralysis then developed in the lower limbs. During the day of September 25, he walked normally, but during the night he arose from his bed at 10 p. m. and found he had difficulty in walking. Shortly after midnight he was unable to move either limb, and sensation in the lower limbs was lost. At the same time he lost control of his rectal and vesical sphincters and became incontinent. The next day he had to be catheterized. Since that time he has remained in the same condition.

September 30, 3 p. m.: A spinal puncture was made in the lumbar region and 10 c.c. of a greenish-yellow fluid were removed. It ran slowly, and before it could be carried to the laboratory it was two-thirds coagulated; when it reached the laboratory it was entirely coagulated. Examination showed a heavy trace of globulin, 30 cells to the cm., equal parts of lymphocytes and leukocytes and no bacilli or other organism. The spinal Wassermann reaction was + + + + in cholesterin. The blood Wassermann reaction was negative. Hemoglobin was 50 per cent.; red blood cells, 4,040,000; white blood cells, 31,200. Roentgen-ray examination revealed nothing of importance.

The face and upper limbs were not affected. Complete anesthesia for all forms of sensation was present anteriorly and posteriorly from about the seventh intercostal space down, including the lower limbs. A line drawn around the trunk at the lower part of the ensiform process gave a zone of hyperalgesia to pin prick. The lower limbs were flaccid and the tendon reflexes of these limbs were lost. Control of bladder and rectum was lost, and a bed sore developed over the sacrum.

On account of the history of a severe boil on the neck in July and xanthochromia of the spinal fluid with signs of a transverse lesion of the spinal cord at about the seventh thoracic root, it seemed evident that some lesion had occluded the dural canal at this level causing the xanthochromia and pressure on the cord, and producing the signs of complete transverse lesion. The severe boil of the neck made the diagnosis of extradural abscess from this boil possible.

A lumbar puncture was made at the tip of the spinous process of the sixth thoracic vertebra, in order to enter the intercostal foramen between the seventh and eighth thoracic vertebrae, and about 6 to 8 c.c. of pus were aspirated at this level. This pus was extradural. Stained preparations showed a staphylococcus. Culture showed pure culture of *Staphylococcus aureus*. The patient was transferred immediately to the surgical wards, and Dr. T. T. Thomas performed a laminectomy at midnight and removed about 2½ drams of pus.

Under autogenous vaccines the leukocyte count of the blood was greatly reduced.

Dr. Spiller, in closing, said that this was at least the third case of this character he had seen, and the subject seemed to be important. The man had the symptoms of a transverse lesion. The lumbar puncture which had been performed showed the Nonne-Froin syndrome. There was clearly obstruction to the circulation of the cerebrospinal fluid. Dr. Spiller believed that pus had entered the vertebral column and gone downward until it met some resistance, and had caused an abscess at a lower level on the outside of the dura but within the vertebral column. There was evidently pressure on the cord, but the lumbar puncture had shown that there was no pus within the spinal dura at the time of the puncture.

NOTE.—Death occurred Oct. 28, 1920. The necropsy performed after the meeting of the society showed that a severe purulent meningitis of the pia of the brain had developed; this probably was of late date. There was also a localized empyema of the right side.

DISCUSSION

DR. T. H. WEISENBURG said that six years ago he had had under his observation a man 67 years of age who had all the symptoms of a spinal cord tumor at the level of the left second, third, fourth and fifth lumbar segments. In addition to pain and diminution of power in both limbs, the patient had also increase of knee jerk on the right, with absence of it on the left. The Achilles' reflex was increased on both sides, and a bilateral Babinski reflex was present. The cremasteric reflex was lost on the left side. Lumbar puncture was made in the second lumbar interspace. It was very painful, and the needle encountered considerable resistance. The amount of fluid removed was considerable, but there were too many blood cells to allow conclusions. Within a week the pain and weakness in the limbs became less, and when he left for home, about a month later, the left knee jerk and cremasteric reflex had returned somewhat and the Babinski reflexes were not so marked. This patient had been kept under constant observation for a period of four years. In a personal communication from his son within a few months, the patient was reported as apparently in good health with no organic symptoms. Since that experience, Dr. Weisenburg had advised and performed punctures at the site of all lesions at which tumors were suspected.

BRACHIAL PARALYSIS FROM THROMBOSIS OF THE SUBCLAVIAN VEIN WITH THE REPORT OF TWO CASES. Presented by DR. GEORGE WILSON.

Dr. Wilson reported the cases for two reasons: first, because of the great rarity of thrombosis of the large veins draining the upper extremities, and second, because one of the patients was thought for a time to have a cerebral monoplegia.

The first case was that of a negro, 25 years of age, admitted to the dispensary for nervous diseases of the Hospital of the University of Pennsylvania, April 12, 1920, and referred to the house the next day. His chief complaint was swelling and paralysis of the right upper extremity. The man stated that he was well until April 12, 1920. On that day he took a nap on a pile of coal ashes still a little warm. He awoke three hours later with numbness in the right arm. He noticed that the entire arm was swollen and that on the extensor surface of the forearm there were several large blisters. He denied syphilis.

At the first examination, because of a suspected weakness of the lower half of the right side of the face, a cerebral thrombosis was considered. The eyes, cranial nerves, heart and abdomen were normal. There was slight enlargement of the posterior cervical glands. The lungs presented evidence of acute bronchitis with infiltration of the right upper lobe. The right upper extremity was greatly swollen from the hand to the shoulder. The swelling pitted on pressure. Radial and brachial pulses could not be felt. On the extensor surface of the forearm and arm were several large blebs. The tendon reflexes were absent and sensation for touch, pain and temperature were distinctly impaired. The entire extremity was completely paralyzed. The other extremities were normal. The sputum, repeatedly examined, was negative for tubercle bacilli. The blood showed a mild secondary anemia with the leukocytes continually below 10,000. A blood culture was sterile. The Wassermann reaction was delayed negative. The urine was negative except toward the end when evidence of nephritis was present. A guinea-pig was injected with a centrifuged sediment of sputum and died from tuberculosis.

The patient recovered some power in the hand and forearm, and the edema disappeared to a large degree. The radial pulse returned with the receding edema. The patient was transferred to the medical service, May 13, 1920.

The diagnosis made was brachial palsy due to a thrombophlebitis of the subclavian vein. The lung condition was considered tuberculous. Death occurred, June 6, 1920. Necropsy examination showed miliary tuberculosis. The mediastinal, cervical and retroperitoneal lymph nodes were caseous. It was unfortunate that the subclavian vein was not dissected out at necropsy.

The second patient shown to the society was a negro, 22 years of age, single, a lead worker. He was admitted to the Philadelphia General Hospital, Sept. 16, 1920, in Dr. Spiller's service. The patient had been admitted to the same hospital three times before; the first time with secondary syphilis, the second time with a bubo and the third time with tertiary syphilis. He worked with lead for some time but quit because of symptoms suggesting lead poisoning.

On July 31, 1920, he worked and went to bed feeling well. He awoke the following morning and found that he was lying on the left arm, that the entire extremity was intensely swollen and that it was paralyzed. He had no pain for the first twenty-four hours, but had much pain after that. He went to a hospital where about thirty incisions were made with the discharge of considerable serum. The swelling subsided to some extent.

The patient was of a low mental type. The eyes, ears, cranial nerves, lungs, heart and abdomen were normal. The left upper extremity was completely paralyzed with lost reflexes and great impairment of sensation from the elbow down. Many scars were present on the arm. The swelling had entirely subsided. There was considerable atrophy of the hand and forearm. Examination of the urine showed lead. Blood examination revealed a secondary anemia with granular degeneration of the red cells. The Wassermann reaction was negative. The diagnosis in this case was the same as in Case 1. In this case the etiologic factor was syphilis, whereas in the first case the cause was tuberculosis.

A considerable degree of recovery has taken place, probably influenced greatly by the early incisions. The patient is on antisyphilitic treatment and is receiving massage and galvanism.

The paralysis in the cases reported was considered due to the pressure of an enlarged and thrombosed subclavian vein on the brachial plexus.

DISCUSSION

DR. CADWALADER said Dr. Wilson's case showed that the brachial plexus could be paralyzed by the pressure exerted on it by a thrombus in the subclavian vein. It was not uncommon to find cases of gunshot wound, in France, in which a hematoma had formed, or a thrombus that compressed the plexus and caused paralysis. When removed or absorbed, the paralysis disappeared.

A CASE OF TABES WITH UNUSUAL SYMPTOMS. Presented by DR. ALFRED GORDON.

A man, 54 years of age, was a typical tabetic. His disease was of many years' standing. About three months before, he developed sharp shooting pains in the midcervical region, radiating laterally. At the same time it was observed that the inner border and lower angle of the left scapula were receding from the thorax when the scapula was at rest. Since the function of the rhomboid muscle is to hold the scapula against the thorax in opposition to the teres major and serratus muscles, it is evident that the left rhomboid was paralyzed. The nerve supply of the latter comes from the fourth and fifth cervical segments. The muscular paralysis and the pain in the midcervical region are indications of a probable extension of the tabetic process to the cervical cord and its meninges.

During the last two years the patient has been suffering also from marked ptialism. The latter was so pronounced that it interfered with sleep. The mouth was always filled with mucus, and the dribbling of saliva was constant. At night the pillow was saturated with it. The involvement of the parotid, submaxillary and sublingual glands was manifest. Their nerve supply comes principally from the sympathetic system. There is, therefore, a sympathetic complication of tabes. Since the amount of mercury used by the patient was extremely small—for the last two years not any—mercury cannot be incriminated in the excessive salivation. The history shows that no other internal factor was the cause of the distressing condition. The condition was probably a tabetic complication.

Book Reviews

DAS WESEN DER PSYCHIATRISCHEN ERKENNTNIS. By ARTHUR KRONFELD, M.D. Pp. 482. Berlin: Springer, 1920.

Not long before the war began, a group of men arose in Germany who were interested in correlating the material of psychopathology with philosophical conceptions. The journal of this group was the *Zeitschrift für Pathopsychologie*. Of this group, Kronfeld has been a conspicuous representative, and this volume initiates a comprehensive treatment of problems presented in this field.

A facile but none the less objective touchstone of a book's content is the index of names. Clearer evidence of the volume's tendency could hardly be offered, within the space limits, than the comparative numbers of times the following writers are indexed: Abderhalden 2, Bergson 12, Bleuler 27, Brentano 42, Brodmann 1, Flechsig 1, Freud 21, Fries 50, Gaupp 1, Griesinger 16, Gudden 1, Hegel 10, Heinroth 10, Husserl 45 (Janet 0), Jung 2, Kant 86, Kraepelin 19, Lipps 47, Monakow 1, Nelson 35, Nissl 2, Pinel 1, Rickert 47, Sommer 3, Wernicke 7, and Wundt 24.

Let this testify to the essentially philosophical orientation of the work, which is so pronounced as to suggest that the psychopathologist should leave any but the most general criticism to someone more practiced in dealing with philosophical concepts. It is hard to see that the practicing psychiatrist is more concerned with it than the geologist with the headstones of a necropolis. Dr. Kronfeld has reared an imposing mausoleum, but most readers of this journal will find the interior tenebrous.

The author gives earnest of more concrete material in work to follow, which may be of more legitimate concern to the psychopathologist. But to him, a book like the present raises somewhat forcibly an underlying question of ideal values. It is a case in which possible benefit to psychopathology or to society seems minute in proportion to the energy systematically and devotedly expended. Purposes of sublimation may have been well served; but not every one's mind endures, St. Antony-like, "ideationizing" so naked and unashamed. Of those more fortunate, there sang that great though here uncited philosopher of our author's tongue,

"Wie wohl ist's dem, der dann und wann
Sich etwas schönes dichten kann!"

LE SYMPATHIQUE ET LES SYSTEMES ASSOCIES: ANATOMIE CLINIQUE, SEMIOLOGIE ET PATHOLOGIE GENERALE DU SYSTEME NEURO-GLANDULAIRE DE LA VIE ORGANIQUE. Par A. G. GUILLAUME. Paper. Price, 6 francs 50 net. Pp. 160, with illustrations. Paris: Masson et Cie, 1920.

This little book of 160 pages deserves the attention of both the internist and the neurologist. It deals with the anatomy, physiology and pharmacology of the visceral nervous system and illustrates these fundamental subjects with simple and readily understandable diagrams. Such subjects as the

anatomy of the different sensory fibers of the visceral system and the distribution of the sympathetic fibers from the thoracico-lumbar cord to the spinal and cranial nerves and thence to the end organs are thoroughly dealt with.

The clinical chapters are brief and, beyond a few observations on the results of trauma occurring from war missiles, contain no new material.

Altogether the book is clearly written, well illustrated and complete from the point of view of anatomy and physiology. As a means of reviewing this subject, it is excellent.

Fifty cents each will be paid for the April and May, 1919, issues of the *ARCHIVES OF NEUROLOGY AND PSYCHIATRY*. Address to American Medical Association, 535 North Dearborn St., Chicago, Ill.

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EPIDEMIC (LETHARGIC) ENCEPHALITIS

CULTURAL AND EXPERIMENTAL STUDIES. PRELIMINARY
COMMUNICATION *

WILLIAM THALHIMER, M.D.

MILWAUKEE

The following report presents evidence believed to be confirmatory of the results of Loewe and Strauss¹ with respect to the filtrability of the virus obtained from cases of epidemic encephalitis and the specific nature of the minute filtrable organism cultivated with special methods by these investigators.

Loewe and Strauss inoculated rabbits and monkeys with material from cases of epidemic (lethargic) encephalitis, following which a large percentage of the animals died, the central nervous system showing typical lesions of encephalitis. The materials injected were blood, spinal fluid, and Berkefeld² filtrates of nasal washings, also Berkefeld filtrates of the central nervous system and nasopharyngeal mucous membranes removed at necropsy. Their results demonstrated an infectious agent in the blood and spinal fluid and in Berkefeld filtrates of these materials from cases of encephalitis. It was found that the infectious agent is a filtrable one. Berkefeld filtrates of the brains of these animals caused the same disease when injected into other rabbits and monkeys. This virus was passed through many series of animals.

They were also able to cultivate on ascitic fluid tissue medium (as perfected by Noguchi³) a minute organism similar in morphologic,

* From the Laboratories of Columbia Hospital. This investigation was carried on with funds generously supplied by the Milwaukee Association of Commerce from a fund collected by them for the purpose of medical investigation. Thanks are gratefully extended to Dr. H. V. Ogden for his stimulating influence during the investigation.

1. Lowe, L., Hirshfeld, S., and Strauss, I.: Studies in Epidemic Encephalitis (Encephalitis Lethargica), *Jour. Infect. Dis.* **25**:378 (Nov.) 1919; *New York M. J.* **109**:772, 1919. Loewe, L., and Strauss, I.: Etiology of Epidemic (Lethargic) Encephalitis, *J. A. M. A.* **73**:1056 (Oct. 4) 1919; The Diagnosis of Epidemic Encephalitis, *J. A. M. A.* **74**:1373 (May 15) 1920; Proceedings of the New York Path. Soc. **20**:18, 1920; Studies in Epidemic (Lethargic) Encephalitis, *Cultural Studies, J. Infect. Dis.* **27**:250 (Sept.) 1920.

2. Mandler clay filters were also used by these investigators.

3. Noguchi, Hideyo: *J. Exper. Med.* **14**:99, 1911.

staining and cultural characteristics to the so-called "globoid bodies" recovered by Flexner and Noguchi⁴ from cases of epidemic poliomyelitis. The organism of Loewe and Strauss is also a filter passer, and cultures of it injected intracranially into monkeys and rabbits produced the typical lesions of epidemic encephalitis in the central nervous system, resulting in death in most of the inoculated animals. The same micro-organism, with the same selective requirements for growth, was recovered from the brains of these animals.

This micro-organism differs from that of Flexner and Noguchi in "virulence, occurrence, and particularly in the ability to infect rabbits." It was also cultivated from the spinal fluid of epidemic encephalitis. Cultures infected rabbits and caused typical microscopic lesions, whereas monkeys were "relatively refractory." Flexner and Noguchi did not succeed in cultivating their organism from the spinal fluid of cases of poliomyelitis, and rabbits could not be infected with it, whereas monkeys were readily infected.

Levaditi and Harvier⁵ also later demonstrated the relationship of a filtrable virus to epidemic encephalitis. With material from cases of this disease, passed through Berkefeld filters, they caused typical lesions in rabbits, monkeys and guinea-pigs, and they passed this virus through many series of animals, but they have not reported the cultivation of a filtrable organism.

McIntosh and Turnbull⁶ inoculated into one monkey an emulsion of pieces of central nervous system from a fatal case of epidemic encephalitis, and into another monkey a Berkefeld filtrate of this emulsion. The first monkey was killed three weeks later when it was in a drowsy condition. The second monkey died after two months, and the central nervous system showed the typical lesions. No cultural studies with ascitic fluid tissue medium were reported.

MATERIALS

This investigation is based on the following material:⁷

1. The central nervous system was obtained from four cases of epidemic encephalitis, two of the fulminating type, one with marked

4. Flexner, Simon, and Noguchi, Hideyo: *J. Exper. M.* **28**:461, 1913.

5. Levaditi, C., and Harvier, P.: *Compt. rend. Soc. de biol.* **83**:354, 1920; *Bull. et mém. Soc. méd. d. hôp. de Par.* **44**:179 (Feb. 6) 1920.

6. McIntosh, J., and Turnbull, H. M.: *Experimental Transmission of Encephalitis Lethargica to a Monkey*, *Brit. J. Exper. Pathol.* **1**:89 (April) 1920.

7. The necropsy material has been secured, through the kindness of Drs. L. M. Warfield and A. G. Margot, from two cases at the Milwaukee County Hospital, and of Drs. A. W. Gray and H. V. Ogden, from cases at Columbia Hospital. The spinal fluids were secured from a patient of Dr. Ogden and from a patient of Dr. Solomon Strouse, at Michael Reese Hospital, Chicago.

myoclonus and one showing the usual form of lethargy. The central nervous system of all had the characteristic lesions described by Marinesco,⁸ Bassoe,⁹ and others, the changes varying, however, in the different cases. The changes included capillary congestion; microscopic hemorrhages; perivascular, mononuclear leukocytic infiltration; microscopic areas of degeneration and necrosis, etc. The two fulminating cases showed marked congestion, great numbers of capillary hemorrhages, a few areas of focalized necrosis with round cell infiltration and here and there slight or marked perivascular round cell infiltration. The other two cases, which ran a longer clinical course, manifested all these lesions, but many, instead of a few, of the vessels were surrounded by round cells. The degree of this perivascular infiltration was also greater.

2. Spinal fluid was secured from one of the patients with a fatal, fulminating case of epidemic encephalitis and from one patient convalescing from a case of epidemic encephalitis of several months' duration, who had manifested marked lethargy and at the time of lumbar puncture still had headache, nausea, vomiting and a slow pulse.

EXPERIMENTS

About 200 rabbits¹⁰ have been inoculated up to the present time. They received intracranially injections through the thin temporal plate, according to the method devised by Loewe. Young, half grown and full grown rabbits were used. About 1 c.c. can be injected by this method into a full grown rabbit, and it is practically always well tolerated.

To prepare the filtrate, pieces from various portions of fresh central nervous system are ground up in a mortar with sterile sand to make a milky emulsion, and filtered through a Mandler clay filter which has previously been found to hold back *B. prodigiosus*.

At necropsy, on such animals the brain is removed aseptically, most of it is fixed in liquor formaldehydi and cut serially into blocks from which sections are prepared. A large piece is placed in the ice chest in 50 per cent. of glycerol; small pieces are placed in culture tubes. The remainder is ground up with sterile sand, passed through a Mandler

8. Marinesco, G.: Contribution to the Study of the Pathological Histology of Encephalitis Lethargica, Reports to the Local Government Board on Public Health and Medical Subjects, New Ser. 121, 1918, London.

9. Bassoe, Peter: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

10. Recently guinea-pigs have also been used, and a number have developed the experimental disease. These results, together with cultural studies on these guinea-pigs, will be reported later.

filter and the filtrate used for inoculation and cultures. Portions of the cervical and lumbar cord are also placed in liquor formaldehydi.

Of the few animals that died soon after the injection, some died from cerebral hemorrhages, and some apparently from the infectious agent injected. Most of the rabbits died in from two to four weeks after inoculation and some as long as ten weeks later. Some were found dead in the morning after apparently having been well the afternoon before. One apparently well animal, while it was being watched, sprang up in its cage, fell down unable to rise and died in five minutes. Another animal died in the same manner when it was lifted from its cage and placed on the floor for observation. Many animals appeared sick in a general way for a number of days before death, were apathetic and had ruffled fur. Some rabbits developed what seemed to be genuine lethargy. They remained quiet and could not be made to walk. Peripheral paralyses developed in some, involving one or more limbs. One rabbit developed inability to swallow, became emaciated in two days, and was killed. One animal showed myoclonic contractions of the head, eyelids and ears, and also had attacks of excitement culminating in marked opisthotonos, followed by periods of calm.

The animals were inoculated with the same material in groups of from three to eight. In some groups 100 per cent. of the rabbits succumbed, in some as low as 50 per cent. They did not all die after the same interval. At times their deaths were several days or weeks apart. In a number of instances two rabbits in the same group would be found dead the same morning.

Two strains of virus from the brains of two fatal cases have been passed through six series of animals, with filtration through a Mandler clay filter after each passage; that is, the Mandler filtrate from the rabbit brain was injected into the succeeding series of rabbits. The other brain and the spinal fluid strains are more recent and have been passed through only one or two series of animals up to date.

The central nervous system of a large number of the rabbits that died appeared more red and congested than normal, and showed typical and marked microscopic lesions¹¹ identical with those found in fatal human cases. These lesions were sometimes widespread and sometimes confined to a small portion of the brain or cord. Many sections must be examined so as not to miss the lesions in some animals. Well developed lesions of all the different types found in human cases were at times present in a single rabbit brain. Often, however, the patho-

11. The lesions produced in animals and those present in human necropsy material will be illustrated in subsequent communications.

logic picture was not as complete as this. Microscopic hemorrhages were sometimes the prominent lesion and only an occasional blood vessel showed perivascular round cell infiltration. The brain lesions of some of the rabbits of subsequent series, injected with the same strain of virus showed, however, focalized necroses, characteristic perivascular infiltration, etc. The cord from some animals showed the same lesions. Occasionally only marked congestion was found throughout.

Several series of animals were inoculated with control specimens of spinal fluid and filtrates of brain tissue passed through Mandler clay filters and obtained from cases entirely unrelated to epidemic encephalitis. These animals were chloroformed after varying periods and the brains were found to be normal.

CULTURAL STUDIES

The ascitic fluid tissue culture medium used is the same as that perfected by Noguchi and used by him in the cultivation of *Spirochaeta pallida*, "globoid bodies" of poliomyelitis and other organisms.

Briefly, it consists of a small piece of sterile kidney from a rabbit, ascitic fluid and inoculum. These are placed in a tall, narrow test tube about 1 by 20 cm. About from 10 to 15 c.c. of ascitic fluid¹² are used, making a column from 10 to 15 cm. tall. This is then layered on top with sterile warm petroleum, which, when it cools and hardens, seals the tube. Each material used is controlled for its sterility by the usual bacteriologic method. The tubes are incubated at the usual thermostat temperature, 37.6 C.

Spinal fluid is inoculated directly. Central nervous system material is ground in a sterile mortar, with sterile sand and normal salt solution, and passed through a Mandler clay filter (tested to hold back *B. prodigiosus*). The filtrates thus obtained are usually practically watery clear.

When brain material has been removed aseptically, as from the rabbits, small blocks of the brain are also placed in separate culture tubes.

Smears of the cultures are made by placing a large drop of the culture on a slide with a 1 c.c. pipet; it is then spread with a platinum loop and dried in the incubator. The smears are placed in pure methyl alcohol for 30 minutes and then placed in ether for three minutes. Following the above process the smears are stained in either Loeffler's methylene blue for two hours or overnight in dilute Giemsa (1 part to 20 parts distilled water). Usually in from five to ten days, in the positive cultures, a slight cloudiness appears at the bottom of the tube

12. Dr. A. G. Margot has kindly furnished large amounts of ascitic fluid from the Milwaukee County Hospital.

about the piece of kidney. This may extend upward a few centimeters and only rarely reaches the top. Marked cloudiness, cloudiness of the entire column of ascitic fluid, or cloudiness more marked at the top, usually means an accidental contamination. Accumulation of gas beneath the layer of petrolatum always means a contamination. Contaminations only occur occasionally, which is of interest, since the technic of making these cultures requires that each tube be opened from four to five times. The cloudiness described does not, however, always appear, and the culture may be positive and still remain clear.

The organism stains a violet-blue with methylene blue. With Giemsa, it stains purplish. The organism is extremely minute and rounded (i. e., spherical) and is from one fourth to one fifth the diameter of a small anhemolytic streptococcus. It is found singly, in pairs, in short chains and in groups. It is uniform in size and stands out sharply with the usual magnification of an oil immersion lens. In the stained smears it must be differentiated from amorphous debris from the piece of kidney, from possible granular precipitate from the stain, etc. It is easily differentiated from ordinary contaminating bacteria. Cultures of this organism have been secured from the Mandler filtrates of the central nervous system of the four fatal cases mentioned and directly from the two spinal fluids. Some of these cultures are now in the sixth successful subculture.

Cultures have been obtained from brain blocks and filtrates in about 80 per cent. of the animals successfully inoculated with the original material. The brains of many animals of the different series, inoculated with filtered virus carried through as many as six series of animals, and the brains of animals inoculated with glycerolated virus also yielded positive cultures.

Five rabbits, inoculated with cultures of the first and third generations of several strains of the organism, showed typical brain lesions, and the same organism was recovered from these brains. The organism was also cultivated from Mandler clay filtrates of various positive cultures.

Cultures have been made from time to time of the various types of inocula on the usual laboratory culture mediums. Rosenow cultures in deep tubes of ascitic fluid glucose agar have been made. Except for occasional accidental contaminations with various kinds of organisms, these control cultures have remained sterile.

Control cultures were made in the ascitic fluid—tissue medium of five spinal fluids received in the laboratory for routine examination, one fluid from a case that proved at necropsy to be a brain tumor and one fluid from a case of acute *Staphylococcus aureus* vegetative endocarditis. Mandler filtrates of the brain from this last case, secured at

necropsy, and also of brains from two monkeys that died from generalized tuberculosis were also cultivated. These control cultures did not show the minute organism described.

SUMMARY

In the experiments cited it is believed that there has been demonstrated an infectious agent which is apparently constantly associated with epidemic encephalitis. These results are identical with those that have been reported by Loewe and Strauss. They are confirmatory of both their animal experiments and cultural studies. These investigators have, in addition, reported successful experiments on monkeys with the same results as in rabbits.

The infectious agent passes through a clay filter capable of holding back ordinary bacteria (*B. prodigiosus*), the filtrate producing a disease in rabbits which is very similar to the clinical features of the human disease. The microscopic brain lesions are identical with those found in fatal human cases, as described by Marinesco, Bassoe and others. These lesions are the same as those described by Loewe and Strauss and illustrated in their publications.

The virus secured from the original sources, consisting of brains from four fatal cases and from two spinal fluids, has been passed through a number of series of rabbits. Two strains have been passed through six series of animals. The brain emulsion was filtered through a Mandler clay filter after each animal passage. Whatever virus was present in the original material would have been diluted out to an infinitesimal amount before the sixth series of rabbits was reached. This demonstrates the infectious agent, or virus, to be of the class usually termed "filtrable viruses."

By means of ascitic fluid tissue medium, as perfected by Noguchi, an extremely minute filtrable organism has been cultivated from the same infectious material. This organism corresponds exactly in cultural characteristics, morphology, staining characteristics and pathogenic properties to that cultivated by Loewe and Strauss.

This organism has been grown from the brains of about 80 per cent. of the rabbits inoculated with filtered virus. It has also been recovered from the brains of rabbits inoculated with cultures of this organism. Only early cultures, up to the third generation, have been injected. Loewe and Strauss have, however, produced the disease with cultures as far removed as the eleventh generation. They proved, by the following experiment, that the small quantity of original virus was so reduced in amount by subcultures that it could not infect animals, and that the virus (i. e., the filtrable organism) must have proliferated:

Two tenths of a cubic centimeter of a filtered virus, which caused typical lesions in animals and which gave positive cultures of the organism described by them, was placed in 15 c.c. of ascitic fluid, exactly as though making a culture. From this dilution 0.2 c.c. was transferred to a second tube of 15 c.c. of ascitic fluid. This was repeated six times. One cubic centimeter of the sixth dilution failed to infect animals when injected intracranially. This demonstrated that even in six transplantations of cultures the original infectious material was so reduced by dilution that the usual dose of 1 c.c. did not infect animals.

It should be noted that epidemic encephalitis appears with different symptoms, varying greatly in type, location and degree. No two cases present the same clinical picture. Also, the microscopic lesions of the central nervous system show corresponding differences in type, degree and location. Similar variations occur in the disease and in pathologic lesions produced in the animal experiments.

Control studies throughout the investigation were uniformly negative.

CONCLUSIONS

1. The experimental and cultural studies of Loewe and Strauss, on epidemic encephalitis, have subsequently been confirmed by the animal experiments of Levaditi and Harvier, McIntosh and Turnbull, and by the experimental and cultural studies reported in this paper.

2. The extremely minute filtrable micro-organism, that has been successfully cultivated in ascitic fluid tissue medium from brain material and spinal fluid obtained from patients with epidemic encephalitis, is believed to be identical with the micro-organism that has been cultivated and reported by Loewe and Strauss.

3. The investigations of Loewe and Strauss, followed by those of Levaditi and Harvier and of McIntosh and Turnbull, taken together with those reported by us, strongly indicate that epidemic encephalitis is caused by a filtrable virus.

4. The filtrable micro-organism cultivated by Loewe and Strauss, and also subsequently cultivated in the course of these studies, is believed to be the etiologic agent of epidemic encephalitis.

JUVENILE TABES

REVIEW OF THE LITERATURE AND SUMMARY OF SEVEN CASES *

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LITERATURE ON JUVENILE TABES

It is natural that the very existence of juvenile tabes dorsalis should be questioned. Among those doubting the reality of such a disease were Marie,¹ von Leyden² and Gumpertz.³ Yet it has now come to be accepted, and the increase in knowledge brought by the Wassermann test and the procedure of spinal puncture has not merely increased the facility of its recognition, but has also widened the scope of our knowledge of its manifestations.

Remak,⁴ in 1885, described the first case of juvenile tabes. Since that time many cases have been reported. On account of the varying standards of what comprises a disease, the literature, up to the time of the recognition of syphilis as a causative factor and of the Wassermann test, was very contradictory. From time to time leading writers have collected series of cases and analyzed them, selecting what in their judgment constituted the picture of juvenile tabes, and rejecting in some cases the very cases on which previous authors had laid greatest stress. Today the literature on the subject may be divided into analyses of collected series and reports of isolated cases. I shall try to give a comprehensive review of the collected series and an analysis of ten isolated cases that I have collected.

Juvenile tabes is a rare disease and quite uncommon as compared with the diffuse form of neurosyphilis of children. It is rare also as compared with juvenile paresis. There are probably ten cases of juvenile paresis to one of juvenile tabes (Jones⁵), and, although adult tabes is more common than adult paresis, the reverse is true in the

*Work done in the Section on Neurology, Mayo Clinic.

1. Marie, P.: *Leçons sur les maladies de la moelle*, Paris, G. Masson, 1892.

2. Leyden, E. von: *Die Tabes dorsalis*, *Real-Encycl. d. ges. Heilk.* **24**:68, 1900.

3. Gumpertz, K.: *Was beweisen tabische Symptome bei hereditär syphilitischen Kindern für die Aetiologie der Tabes?* *Neurol. Centralbl.* **19**:803, 1900.

4. Remak, B.: *Drei Fälle von Tabes im Kindesalter*, *Berl. klin. Wchnschr.* **22**:105, 1885.

5. Jones, E.: *The Symptoms and Diagnosis of Juvenile Tabes*, *Brit. J. Child. Dis.* **5**:131, 1908.

juvenile forms. In spite of its rarity, however, we no longer doubt that there is a definite disease called juvenile tabes, which may produce any one or every one of the signs and symptoms of adult tabes.

There seems to be no great predilection for either sex in the juvenile form, whereas in the adult form the predatory male more often acquires syphilis and later tabes. Females seem slightly to predominate in the juvenile forms; in three series there were fifty-eight girls to forty-five boys. In Cantonnet's⁶ series 62 per cent. were girls and 37 per cent. boys.

With regard to the age incidence, account must be taken as to whether the syphilis is congenital or acquired. In the acquired cases, it is easy to understand that syphilis must be acquired early to produce juvenile tabes. In a case Marburg⁷ records, the child was infected at the age of 2 by a nurse and developed tabes at 8, the youngest patient with tabes due to acquired syphilis on record. In the cases of congenital syphilis, by far the greatest number in the various series, the age incidence was as follows:

In the Hirtz and Lemaire⁸ series the average age was 14.3; in Cantonnet's series the average age was 15, and in the series of ten cases that I collected the average was 14. The average age is, therefore, about 14, or puberty. The youngest of the patients was one of Mingazzini and Baschieri Salvadori.⁹ The child was 3 years old when symptoms began. The youngest in Hirtz and Lemaire's series was 6. Bertolotti¹⁰ reported a case of tabes due to inherited syphilis in which the patient was 40. That syphilis is the chief factor in the etiology of the disease is now accepted, but to trace its origin in some of the cases is interesting, considering how much in doubt the older writers were as to the part it played in the disease. With our modern methods for the detection of the disease, we do not lay so much stress on the absence or the presence of a history of parental syphilis as did early authors. The parental origin of infantile syphilis is now regarded as demonstrated when the whole family gives a total inhibition Wassermann test, although the history reveals no trace of acquired syphilis in the parents. We may suspect that it is a case of acquired juvenile

6. Cantonnet, A.: Les manifestations oculaires du tabes juvenile, *Arch. d'ophth.* **27**:708, 1907.

7. Marburg, O.: Klinische Beiträge zur Neurologie des Auges. Infantile und juvenile Tabes, *Wien. klin. Wchnschr.* **16**:1295, 1903.

8. Hirtz, E., and Lemaire, H.: Etude critique sur le tabes infantile juvenile, *Rev. neurol.* **13**:265, 1905.

9. Mingazzini, G., and Baschieri-Salvadori, G.: Considerazioni cliniche sulla tabe ereditaria, *Riv. di patol. nerv.* **11**:580, 1906.

10. Bertolotti, M.: Tabes ereditaria tardiva per sifilide congenita, *Riforma med.* **21**:93, 1905.

tabes when the tests of both parents fail to show evidence of syphilis. In a case in the records of the Mayo Clinic a child had all the signs of juvenile tabes, but her supposed mother, who was being examined at the same time, had no signs of syphilis, including a negative Wassermann test. The child proved, after some inquiry, to be an adopted daughter of doubtful parentage. Nonne¹¹ reports a child infected at the age of 5 through sleeping with a lodger. The child conveyed the disease to both parents by kissing; finally all three developed tabes. Nonne also tells of a mother who infected herself wet nursing an infant, and then infected her own child, who later developed juvenile tabes.

The number of recorded cases of juvenile tabes due to acquired syphilis is relatively small compared with the number due to hereditary syphilis. In Lasarew's¹² twenty-three cases only two were recorded as due to acquired syphilis, and in Hirtz and Lemaire's series of forty-six cases of juvenile tabes syphilis was acquired early in childhood in only three. A patient to become tabetic while still juvenile, that is under 21, must necessarily receive the primary lesion at an early age. Von Halban¹³ recorded a case in which the child at 4 months of age was infected with syphilis from a nurse; later it developed juvenile tabes. Kutner¹⁴ described a case in which the child was infected at 5 by the kiss of a prostitute.

The question of specificity of spirochetes that produce types of diseases of the central nervous system as compared with the influence of the soil they settle on enters into this disease. Is there a particular strain of spirochetes which produces tabes or general paresis in a person, or do the results of infection depend on the type of his nervous system or his family predisposition, or, in short, on some condition for which he is responsible irrespective of the strain of the spirochetes? This is partially answered by the characteristics of juvenile tabes. The frequency with which the parents of juvenile tabetic patients are either paretic or tabetic stands out. Cantonnet, in his series, showed that in 15 per cent. one parent was tabetic and in 12 per cent. paretic.

11. Nonne, M.: *Syphilis und Nervensystem*, Berlin, Karger, 1902; *Ueber die Bedeutung der Syphilis in der Aetiologie der Tabes*, *Fortschr. d. Med.*, No. 29:986, 1903; *Ein Fall von familiärer Tabes dorsalis auf syphilitischer Basis. Tabes bei der Mutter und ihren zwei hereditär-syphilitischen Töchtern*, *Berl. klin. Wchnschr.* 41:845, 1904.

12. Lasarew, W.: *Ein Beitrag zur Tabes in jungem Alter (Tabes infantilis und juvenilis)*, *Neurol. Centralbl.* 24:988, 1905.

13. Halban, H. von: *Ueber juvenile Tabes nebst Bemerkungen über symptomatische Migraine*, *Jahrb. f. Psychat. u. Neurol.* 20:343, 1901.

14. Kutner, R.: *Ueber juvenile und hereditäre Tabes dorsalis*, *Inauguration Dissertation*, Breslau, 1900.

Nonne records a case in which a sister, as well as the mother, had tabes, also the case of a brother and sister with tabes, and an instance of twin brothers developing tabes at the same age. Grinker¹⁵ reports a family in which the father was syphilitic, the mother tabetic, one son paretic and a daughter hemiplegic. Smith¹⁶ also reported a family of four; the father had paresis, the mother Argyll Robertson pupils, one son juvenile paresis, and one daughter juvenile tabes. Since these few cases were reported many more have been recorded, showing how frequently a family of four or five becomes affected with the same type of neurosyphilis. In some cases a definite neuropathic family tendency can be demonstrated which seems to be a suitable soil for the production of tabes or paresis. On the other hand, it has been shown again and again that paresis or tabes, or both, may be produced in several persons by infection from the same source. The question of the relative importance of a neuropathic heredity and of a possible specific syphilitic nerve virus is yet to be settled.

The course of juvenile tabes is always long and protracted. Juvenile tabes is never acute or fulminating, as adult tabes is occasionally. All observers who have had the opportunity of following the cases for long periods agree to this. Mendel,¹⁷ in 1895, described a case a report of which was first published by Remak in 1885, then by Hildebrand¹⁸ in 1892. In these ten years the progress of the disease was exceedingly slight. Two things follow from this chronicity: First, cases in adults are likely to be diagnosed as acquired tabes unless a careful history is taken; and second, necropsies in these cases are few because in the lengthy course of the disease the patients drift away from observation. Cantonnet followed the histories of twelve patients from ten to twenty years; there was not a death during that time. Large numbers of juvenile tabetic patients, however, drift into taboparesis; even larger numbers become paretic and asylum inmates, and the final change clouds the initial tabes. The rare recognition of juvenile tabes is partly explained by the later change.

No symptom that occurs in adult tabes may not occur in the juvenile form (Skala¹⁹). The symptoms predominate in a different manner, however; some are more common in juvenile than in adult tabes, and

15. Grinker, J.: A Case of Juvenile Tabes in a Family of Neurosyphilitics, *J. Nerv. & Ment. Dis.* **31**:753, 1904.

16: Smith, H. J.: Presentation of a Tabetic Child and Paretic Parents and Brother, *J. Nerv. & Ment. Dis.* **44**:66, 1916.

17. Mendel, E.: Die hereditäre Syphilis in ihren Beziehungen zur Entwicklung von Krankheiten des Nervensystems, *Festschr. f. G. Lewin, Beitr. z. Dermat. u. Syph.* 1896, p. 138.

18. Hildebrand: Quoted by Jones.

19. Skala: Quoted by Jones.

some of the commoner symptoms in the adult disease are rarely seen in the juvenile. Vesical disturbance is most often incontinence, either diurnal or nocturnal, in the latter case not to be distinguished from the common bed wetting of infants. In the order of frequency of symptoms, it is the most constant and earliest and occurred in nearly half of the cases in Hirtz and Lemaire's series and was initial in twelve twenty-eighths. Dydynski²⁰ gave it equal importance with optic atrophy. Marburg found it in twenty of thirty-four of his cases, and Lasarew in fourteen of twenty-three. Lasarew found it in the initial symptom in seven of twenty-three. In the series of isolated cases which I collected, it occurred in five of ten, 50 per cent. The average percentage of all series is 55.

Ocular symptoms follow incontinence in frequency, and in some series equal vesical disturbances in their constancy and frequency as initial symptoms. The first symptom was amblyopia due to optic atrophy in 14 per cent. of Cantonnet's cases. The same percentage occurred in Hirtz and Lemaire's series. As a definite finding it was present as follows: Cantonnet's series, 43 per cent. of 89 patients; Hirtz and Lemaire's series, 36 per cent. of 46 patients; Marburg's series, 33 per cent. of 34 patients; collected cases, 70 per cent. of 10 patients; average, 45 per cent. Other ocular findings, such as Argyll Robertson pupils were present in 87 per cent. of Lasarew's cases, in 73 per cent. of Marburgs, in 73 per cent. of Hirtz and Lemaire's and in 80 per cent. of the series I collected; the average is 78 per cent. This is practically the same percentage as in adult tabes. Irregularity and inequality of the pupils occur with about the same frequency. Ocular palsies and diplopia, if they occur at all, are late symptoms (Jones).

Lightning pain was the initial symptom in 25 per cent. in Cantonnet's series, and in Hirtz and Lemaire's series. They were present in Lasarew's series in 91 per cent., in Marburg's series in 71 per cent., in Hirtz and Lemaire's series in 32 per cent., and in my collected cases in 10 per cent. The average percentage was 51. Jones believes that after explaining the discrepancy by the difference of opinions of authors as to which cases shall be termed juvenile tabes, the number of patients affected by lightning pains is about the same in both types of tabes, that is, 88.4 per cent.

Headache has been included by Jones as a common symptom. Von Halban laid great stress on the appearance of migraine early in the disease. Girdle pains are rarer in the juvenile form than in the adult

20. Dydynski, L. von: *Tabes dorsalis bei Kindern, nebst einigen Bemerkungen über Tabes auf der Basis der Syphilis hereditaria*, Neurol. Centralbl. 19:298, 1900.

form. They occur in only 16 per cent. of the total number of Lasarew's and Marburg's cases.

Sensory changes were present in 65 per cent. of Hirtz and Lemaire's series, in 91 per cent. of Lasarew's cases, in 91 per cent. of Marburg's cases, and in 30 per cent. of the cases I collected, a fairly large discrepancy between the series, which gives an average of 68 per cent. that must be accepted with caution.

All writers agree that the most striking phenomenon in the adult form of the disease, the ataxia which Duchenne so carefully described, is relatively rare in juvenile tabes. Marburg estimates it as occurring in 33 per cent. of the cases, Linser,²¹ in 33 per cent., Lasarew in 63 per cent. and Hirtz and Lemaire in 4 per cent.; in my series it occurred in 20 per cent. This gives an average of 30 per cent. and contrasts remarkably with the series in the adult type in which the frequency has been estimated as 66.6 per cent. Roughly, only one third of juvenile tabetic patients are ataxic, while twice that number of adult tabetic patients have difficulty in locomotion. Cantonnet states that the difficulty is unusual, and he explains it as being due to optic atrophy. Dydynski states that ataxia is either absent or late in appearing in juvenile tabes. The fact that this symptom is rare does not preclude the possibility of its being present and present early (Jones).

Gastric crises are not unknown in juvenile tabes; they are not even rare; 17 per cent. of the patients of Lasarew's series complained of them, 20 per cent. of Marburg's, and 19 per cent. of Hirtz and Lemaire's, giving an average of 18 per cent. thus affected.

Trophic disturbances have been recorded. Nonne has described a case in which a juvenile tabetic patient developed a Charcot joint. Romberg's sign was seen in 75 per cent. of the series analyzed and Westphal's sign was found in about 90 per cent. In the series I collected the patellar reflexes were absent in 80 per cent.

From the foregoing survey of these series of cases of juvenile tabes, we are now in a position to draw some conclusions with regard to the relative frequency of the different symptoms and their order of appearance, and to compare them with the symptoms in adult tabes. Most remarkable is the frequency of vesical disturbance and amblyopia, both as initial symptoms and as constant features of the disease. Hirtz and Lemaire call attention to the insidious manner in which the disease appears, how often the oculist, after discovering early optic atrophy in a child thought of testing the patellar reflexes and was first to diagnose a tabes that had not been ushered in by such striking symptoms as ataxia or lightning pains. In 16.66 per cent. of the Hirtz-Lemaire

21. Linser, P.: Ueber juvenile Tabes und ihre Beziehungen zur hereditären Syphilis, München. med. Wehnschr. 1:637, 1903.

series incontinence and slight sensory changes were the only symptoms calling attention to the disease, and it was only after finding Westphal's sign and Argyll Robertson's pupils that a suspicion arose as to the nature of the disease.

So much for the gleanings from literature up to 1908, when our knowledge of neurosyphilis was not so complete as it is now. In those days when diagnosis was made chiefly by symptoms and physical signs, so insidious a disease as juvenile tabes was overlooked first, because its relation to syphilis was still viewed with suspicion, and second, because its syphilitic origin granted, there was no Wassermann test to fill in the gaps left by the absence of stigmas or family history of the disease. Just because so striking a sign as ataxia was rare, its existence was doubted, and worse still, those who accepted it as an entity were ready to confound it with Friedreich's ataxia and cerebellar ataxia and to write articles on cases they imagined were types of the disease. Whereas in the latter part of the twentieth century ataxia was the deciding point in the diagnosis, now a definite finding of syphilis is demonstrated. The general accumulation of data has shown the existence of the disease, shown the differences, and raised the question as to why there should be differences. I believe that an infection implanted on a tissue that has not yet fully developed ought to produce a different result from that obtained when the same infection is sown on a fully differentiated and completed nervous system. In the selection of the cases of juvenile tabes from the records of the Mayo Clinic, I have guided myself by giving most importance to (1) definite evidence of syphilis, (2) presence of Argyll Robertson pupils, (3) optic atrophy, (4) Westphal's sign, or absent tendo-achillis reflexes, (5) incontinence of urine, and (6) absence of all mental deterioration. The case will be described in the order of the patients' admission to the Clinic.

REPORT OF CASES

CASE A151370.—R. M., a young man, aged 17, came to the Clinic Feb. 3, 1916, complaining of vomiting that was without any relation to meals. He gave a typical history of gastric crises, beginning at the age of 11. Examination showed a striking condition of hypotonia and such laxity of the joints that the patient could twist his body and limbs into most grotesque postures. There were Hutchinson's teeth, a saddle shaped nose, external strabismus, and unequal pupils; Argyll Robertson pupils, Westphal's sign, no Achillis' tendon reflex, and some diminution of pain and temperature sensation over the forehead and feet. No Romberg sign, ataxia, or incoordination was present. The Wassermann test on the blood was strongly positive. Examination of the spinal fluid was negative.

CASE 205051.—E. S., a boy, aged 7, was brought to the Clinic Aug. 15, 1917. For one year before examination the child had had frequent attacks of vomiting, lasting from three to six days, and ending suddenly. He felt well between

attacks and ate to make up for the loss during attacks. He urinated involuntarily and had difficulty in starting the stream. Examination showed general adenopathy and carious teeth. The pupils did not react to light or during accommodation; Westphal's sign was present, the tendo-achillis reflex was diminished, and sensation of pain was diminished over the third dorsal segment. Abadie's sign and some hypotonia were present; there was no Romberg's sign, ataxia, or incoordination. The Wassermann test on the blood was positive, also in the father and mother.

CASE 241224.—K. H., a boy, aged 14, came to the Clinic Aug. 6, 1918, complaining of blindness, which had begun four years before; at the time of examination he was blind in the left eye and could see only 2/200 in the right eye. He had had lightning pains during the last two years. Examination showed an early saddle nose and Hutchinson's teeth. The left pupil was larger than the right, and both were irregular. Reflexes to light and during accommodation were absent. There were complete primary optic atrophy of both nerve heads, hypotonia of all the muscles, and diminished sensation of pressure and vibration over both lower extremities. Westphal's sign and diminished tendo-achillis reflex were present. Examination of the spinal fluid showed a strongly positive Wassermann reaction with 0.3 and 0.4 c.c., a positive Nonne reaction, and 83 small lymphocytes. No Lange test was performed. He gave a strongly positive Wassermann reaction on the blood.

CASE 303391.—C. V. S., a boy, aged 15, presented himself Jan. 20, 1920, complaining of blindness. Seven years before he could see well; since that time sight had been failing gradually, and he was able to distinguish only light and dark. His pupils reacted neither to light nor during accommodation; there were simple optic atrophy of the right eye and an interstitial keratitis of the left. Examination showed a strongly positive total inhibition Wassermann test of his blood. Westphal's sign was present, and the tendo-achillis reflexes were absent. Some slight incoordination was found, and Romberg's sign was present. There was diminution of pain sensibility over the arms and legs. Vibration sensibility was diminished in the lower extremities. The spinal fluid examination showed a negative Wassermann and Nonne test and 36 small lymphocytes.

CASE 305100.—P. F., a young woman, aged 19, presented herself for examination Feb. 4, 1920, complaining of blindness and headaches. She had had frontal headaches since the age of 12, and failing vision in both eyes for the last four years until she barely had light perception. The left eye failed first. Her gait had been staggering and uncertain, and for the last eighteen months she had had pains in the calves corresponding to lightning pains. She had received five intracranial arsphenamin injections through a trephine hole in the skull; but there had been no improvement. Examination showed simple primary optic atrophy complete in both optic disks. Pupil reflexes were absent. There was a diminution in the vibration sense in both upper and lower extremities. Joint sensibility was almost absent in both feet. Westphal's sign was present and the tendo-achillis reflexes were absent. There was great incoordination in the heel and knee test; the gait was extremely ataxic and the Romberg sign was positive. The Wassermann test of the blood was negative, but the spinal fluid showed a total inhibition Wassermann with 0.3 and 0.4 c.c., a negative Nonne reaction, 306 lymphocytes and 32 polymorphonuclears.

CASE 306325.—N. N., a young woman, aged 19, presented herself for examination Feb. 16, 1920, complaining of pain in the neck, shoulder and occiput, which

had commenced fifteen years before, frequent and severe sore throat and insomnia. Examination showed convergent strabismus of the right eye, irregular and Argyll Robertson pupils. There was slight incoordination of the hands, Westphal's sign was present and the tendo-achillis reflex was absent. Vibration sense over both malleoli was diminished; there was no Romberg sign, no ataxia, no incoordination. The Wassermann test of the blood serum was strongly positive with total inhibition, while the Wassermann test with 0.3 and 0.4 c.c. of the spinal fluid was strongly positive also with total inhibition. There was a negative Nonne reaction, 240 small lymphocytes, and a "syphilitic curve" of 1112221000 in the Lange test.

CASE 266511.—G. B., a boy, aged 12, was admitted to the hospital April 3, 1919, complaining of pains in the joints of the wrists, elbows and knees, and of attacks of abdominal pain; the joints were swollen and tender. At times the patient could not hold his urine. Examination showed no hereditary stigmas, but the blood gave a positive Wassermann test with total inhibition. One pupil was much larger than the other and both were irregular. Argyll Robertson's pupils and Westphal's sign were present. Both tendo-achillis reflexes were absent, but the Romberg test was negative. There was no ataxia or incoordination. The boy was seen a year later, but, although examination showed a complete cessation of joint trouble, all other findings were the same; he complained of shooting pains on the outer side of his legs. The spinal fluid was negative.

CONCLUSIONS

To draw any sweeping conclusions from so small a number of cases would invite error; however, something may be learned from their study.

In all seven cases Westphal's sign was present, in six some degree of diminution of sensibility was found, in four the pupils were immobile, in three Argyll Robertson pupils were present, in three optic atrophy, in three crises, in three lightning pains, in three incoordination, in three signs of congenital syphilis, or syphilis outside the central nervous system, in three hypotonia, and in two incontinence of urine. In one only was ataxia present. The serum Wassermann test was triple positive in all but one case. In six cases a spinal fluid examination was made. In half that number the Wassermann test was negative. One of three cases in which the spinal fluid Wassermann test was negative had a cell count of 36. A Lange test was performed once, only, and that had a "syphilitic curve." The patient who gave a negative serum Wassermann reaction also gave a total inhibition spinal fluid Wassermann reaction. Without drawing conclusions, it is possible to remark on (1) the frequency of some pupillary change, (2) the frequency of sensory changes, (3) the equal frequency of optic atrophy, crises, lightning pains, and incoordination, and (4) the relative infrequency with which incontinence of urine and ataxia were encountered, especially ataxia. It seems remarkable that in the six spinal fluids examined, three should have been negative in spite of well-marked symptoms and positive blood serum.

Hypotonia, a phenomenon not mentioned in any of the literature I read, was present in three cases, and in one case was the most striking feature. At the same time, children are much more flexible than adults, and how much is normal or how much abnormal for a child is hard to determine.

Another noticeable feature is that of seven patients with disturbances of pupil reflexes; four had no movement to light or during accommodation. The large immobile irregular pupil is more frequent with juvenile tabetic patients in contrast to the myosis and Argyll Robertson pupil of adult patients.

In the comparison of my findings in these few cases with those of former authors there is little essential difference, except that I found vesical incontinence not so frequent as reported. Perhaps if a larger number of cases had been at hand, my percentage would have been larger. The cases agree well with regard to the paucity of physical signs, the long insidious course and the rarity of ataxia. The Wassermann tests of the serum and spinal fluid have simply made the diagnosis easier.

SUMMARY

Juvenile tabes is now a well recognized entity, and with our increased facility of diagnosis, more and more cases are being recognized and reported. The features of the disease that I have attempted to show may be briefly summarized: The insidious onset, the lengthy and even latent course of the disease, during which time no subjective complaint may be made, are in contrast to the frequent optic atrophy and total blindness that may also occur. The frequency of incontinence of urine, the relative rarity of such striking phenomena as ataxia, girdle sensations and lightning pain, and finally the frequent parietic termination, are features that stamp juvenile tabes with a distinctive mark and distinguish it from the adult type.

INTERPRETATION OF THE "PARETIC CURVE" IN LANGE'S COLLOIDAL GOLD TEST*

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The occasional occurrence of a paretic gold curve in cases that were not paresis, and the occasional diagnosis of paresis without a paretic gold curve suggested that a closer study of this particular colloidal gold reaction should be made. A review of the rather extensive literature that has grown up since Lange¹ first introduced this test in 1912 shows that various conclusions have been drawn concerning the value of the paretic curve. These conclusions, however, were based on a more or less limited number of cases.

At this hospital about 8,400 colloidal gold tests have been made on about 7,100 different cerebrospinal fluids. Of this number, 677 fluids gave paretic curves, and these constitute the basis for this study. A large majority of the patients coming to this hospital are in the early stages of their disease, and consequently these findings are those of the early stages, and are, therefore, the more valuable. Another advantage is that in almost every instance a fluid is obtained before antisyphilitic treatment is started. The diagnoses of all the cases considered have been confirmed by a long period of observation at this or some other state hospital, and in addition many have been confirmed by necropsy.

The preparation of the colloidal gold, the technic of the test, and the reading of the results have all been adequately described by many writers;² these points will, therefore, not be taken up in this article. I shall consider only the interpretation of the paretic gold curve.

* From the Massachusetts State Psychiatric Institute, Boston.

1. Lange: Ueber die Ausflockung von Gold Sol durch Liquor Cerebrospinalis, Berl. klin. Wchnschr. **49**:897, 1912,

2. Lange: Ueber die Ausflockung von Gold Sol durch Liquor Cerebrospinalis, Berl. klin. Wchnschr. **49**:897, 1912. Kaplan: Serology of Nervous and Mental Diseases, Philadelphia, W. B. Saunders Company, 1914. Miller, Brush, Hammes and Felton: A Further Study of the Diagnostic Value of the Colloidal Gold Reaction Together with a Method for the Preparation of the Reagent, Bull. Johns Hopkins Hosp. **26**:391, 1915. Warwick and Nixon: A Study of the Colloidal Gold Reaction and Its Clinical Interpretation, Arch. Int. Med. **25**:119 (Feb.) 1920. Lowrey: Cerebrospinal Fluids, Especially the Gold Reaction in Psychiatric Diagnosis, J. Nerv. & Ment. Dis. **46**:186 (Sept.) 1917.

First, what shall be called a paretic curve? It is agreed that the typical paretic curve gives the following reading: 5555543100,³ but there are many variations that may still be called paretic curves. The number of tubes showing complete precipitation may be increased or decreased somewhat without altering the interpretation. In one case of undoubted paresis after an acute exacerbation and after some intraspinal treatment, all the usual ten tubes were reduced to fives with a sudden drop to the normal color in further dilutions. This case had previously given typical paretic curves, and soon after the typical curve reappeared. It also seems from experience with other cases that the number of fives may be reduced to two or three and the result may still be called a paretic curve if the general form of the typical curve is present. A reading such as 4444432100 should certainly be called a paretic curve, especially when the fours are well marked. However, if the reading starts with only threes, we are on the borderline between the paretic and the syphilitic curve. Therefore, if the general conformation of the typical paretic curve is present, and the reduction in the first three or more tubes is well marked in the fours or is in the fives, we may consider it a paretic curve. An irregular reading, such as 4525231000, may be found after intensive treatment, as a result of poor gold solution or in contaminated fluids, and such a reading should always be questioned.

GENERAL CONSIDERATIONS

Because of the name, "paretic curve," the question is often asked, "Does a paretic curve always mean paresis?" or in other words, "Is it pathognomonic of paresis?" This question has been answered in the negative by many previous writers. Of this series of 677 paretic curves, thirty-nine cases were not paretic; of these fourteen were not cases of neurosyphilis (Table 1).

PARETIC CURVES IN PARESIS

Another question often asked is whether or not the fluids from cases of paresis always give paretic curves. This can also be answered in the negative by the fact that of 638 cases of paresis, a paretic curve was not found in the first fluid tested in eleven cases. However, four of these later developed a typical curve, and in the remainder only one specimen of spinal fluid was examined.

Among the cases of paresis just mentioned are included the cases diagnosed as taboparesis and juvenile paresis. These types gave typical results in every instance but two. A few other cases were found in

3. Solomon, Koefod and Welles: The Diagnostic Value of Lange's Gold Sol Test, Boston M. & S. J. **173**:957, 1915.

TABLE 1.—GENERAL DISTRIBUTION OF PARETIC CURVES

Author	Number of Paretic Curves	In Paresis		Cerebrospinal Syphilis or Tabes		In Diseases Other Than Neurosyphilis	
		Number	Percentage	Number	Percentage	Number	Percentage
Miller, Brush, Hammes and Felton ⁴	151	129	85.4	19	12.5	3	2.1
Lowrey ⁵	69	65	93.6	3	4.3	1	2.1
Swalm and Mann ⁶	62	62	100.0	0	0.0	0	0.0
Rawlings ⁷	102	101	99.0	0	0.0	1	1.0
Hammes ⁸	52	42	80.7	9	17.3	1	2.0
Eicke ⁹	50	50	100.0	0	0.0	0	0.0
Jaeger and Goldstein ¹⁰	48	33	68.7	12	25.0	3	6.3
Eskuchen ¹¹	18	17	94.4	1	5.6	0	0.0
Cases in this series	677	638	94.2	25	3.7	14	2.1

4. Miller, Brush, Hammes and Felton (Footnote 2).

5. Lowrey (Footnote 2).

6. Swalm and Mann: New York M. J. **101**:719, 1915.

7. Rawlings: The Colloidal Gold Reaction in Four Hundred and Ninety-Eight Psychiatric Cases, Arch. Neurol. & Psychiat. **2**:180 (Aug.) 1919.

8. Hammes: The Comparative Value of the Wassermann, the Colloidal Gold and Other Spinal Fluid Tests, Am. J. M. Sc. **154**:625 (Nov.) 1917.

9. Eicke: Die gold reaktion im liquor cerebrospinalis, München. med. Wchnschr. **60**:2713, 1913.

10. Jaeger and Goldstein: Goldsolreaktion im liquor cerebrospinalis, Ztschr. f. d. ges. Neurol. u. Psychiat. O. **16**:219, 1913.

11. Eskuchen: Die fünfte reaktion, Ztschr. f. d. ges. Neurol. & Psychiat. O. **25**:486, 1914.

TABLE 2.—CASES OF PARESIS WITHOUT A PARETIC CURVE IN THE FIRST FLUID TESTED

Number	Date	Colloidal Gold Curve	Glucose	Albumin	Cell	Wassermann Test		Remarks
						Cerebrospinal Fluid	Serum	
6941	6/24/16	0001232100	3	3	58	55000*	Positive	Taboparesis. On admission showed typical symptoms of paresis
	7/25/16	5555533000	3	3	24	55500	Positive	
11514	10/29/18	2223310000	0	N	0	Positive	Negative	Diagnosis of paresis recently confirmed by necropsy
	11/12/18	5554443310	0	1	1	Positive	Positive	
4303	11/17/15	3333210000	2	2	55	Doubtful	Positive	Typical paretic symptoms
9217	8/16/16	3333211000	1	1	27	Positive	Positive	Cause of death, general paralysis
12036	1/27/19	0004521000	1	1	2	Positive	Positive	Cause of death, general paralysis
11123	8/10/18	1112321000	2	2	116	50000	Positive	Diagnosis, taboparesis; treated since first L.P.
	12/ 2/18	5554443221	1	1	2	555540	Positive	
	11/ 2/19	5555443100	1	1	2	555550	Positive	
9496	10/14/17	3332100000	3	3	27	Positive	Positive	Diagnosis confirmed at Taunton State Hosp.
11981	1/17/19	0001131100	1	2	17	Positive	Positive	
9244	8/21/17	3333210000	1	1	46	Positive	Positive	Diagnosis confirmed at Westboro State Hosp.
8520	4/18/17	1132210000	0	2	38	Positive	Positive	
7788	12/ 6/16	2221000000	3	4	59	55555	Positive	Treated after first L.P.
	12/23/16	5555443300	3	3	40	50000	Positive	
	1/24/17	1112310000	3	4	23	55555	Positive	

* The reaction is recorded in various dilutions of spinal fluid from 2 c.c. to 0.1 c.c. The figure 5 means a strongly positive reaction.

which there was not a paretic type of curve, but all of the patients had received previous intensive treatment before they were examined at this hospital.

The objection may be made that few cases were diagnosed as paresis in the absence of a paretic curve. That is the tendency, but it was found that many cases were diagnosed as paresis without a paretic curve; most of them, however, were later proved to be incorrectly diagnosed. One case, for instance, was consistently diagnosed as general paralysis in spite of the absence of a paretic curve in several fluids tested, but at necropsy no evidence, either microscopic or macroscopic, could be found to support this diagnosis.

TABLE 3.—PARETIC CURVES IN PARESIS

Author	Number of Cases	Colloidal Gold Curve								Wassermann Test	
		Paretic		Syphilitic		Atypical		Negative		Percentage Positive	Percentage Negative or Doubtful
		Number	Percentage	Number	Percentage	Number	Percentage	Number	Percentage		
Lowry ⁵	94	69	73.4	7	7.4	16	17.0	2	2.2	88.3	9.7
Eskuchen ¹¹	21	17	81.0	3	14.0	1	5.0	0	0.0	62.0	38.0
Hammes ⁸	43	42	98.0	1	2.0	0	0.0	0	0.0	96.0	4.0
Eicks ⁹	52	50	96.0	0	0.0	0	0.0	2	4.0	96.0	4.0
Larkin and Cornwall ¹²	99	86	87.0	—	—	—	—	—	—	95.0	5.0
Rawlings ⁷	120	101	84.0	7	6.0	11	9.0	1	1.0	96.0	4.0
Swalm and Mann ⁶	70	62	89.0	3	4.0	4	5.7	1	1.3	92.8	7.2
Miller and Levy ¹³	49	49	100.0	0	0.0	0	0.0	0	0.0	—	—
Miller, Brush, Hammes and Felton ⁴	130	126	96.9	1	1.0	2	2.0	2	2.0	93.0	7.0
Weston ¹⁴	34	31	91.0	3	9.0	0	0.0	0	0.0	91.0	9.0
Jaeger and Goldstein ¹⁰	33	33	100.0	0	0.0	0	0.0	0	0.0	94.0	6.0
Present series.....	638	627	98.2	8	1.2	3	0.6	0	0.0	96.4	3.6

12. Larkin and Cornwall: Value of Laboratory Diagnosis in Neurosyphilis, *Am. J. Syphilis* **3**:76, 1919.

13. Miller and Levy: Colloidal Gold Reactions in the Cerebrospinal Fluid *Bull. Johns Hopkins Hosp.* **25**:133, 1915.

14. Weston: Colloidal Gold and Other Tests Applied to the Spinal Fluid, *Am. J. Insan.* **71**:773, 1914.

PARETIC CURVES IN OTHER DISEASES

Cerebrospinal or Diffuse Neurosyphilis.—The differentiation of cerebrospinal syphilis from paresis is difficult, especially in the early stages, and apparently the colloidal gold test is not to be relied on entirely when such a question arises. In cerebrospinal syphilis the syphilitic type of curve (1233210000 or 3332110000) is most often found. However, in the series of cerebrospinal syphilis cases at this hospital several paretic curves were found in the first fluid examined, and some fluids developed a paretic reaction later. No case of this type was considered unless it was so typical that paresis could be

excluded entirely. Of thirty-one cases of cerebrospinal syphilis, fifteen gave a paretic curve in the first fluid tested and six gave syphilitic curves; in four the colloidal gold curve was negative, and the remaining six gave atypical readings. Of the fifteen cases with paretic curves, all but three showed marked psychotic symptoms, while those with the milder reaction showed chiefly physis symptoms, such as cranial nerve palsies, and the mental symptoms were more or less in the background or entirely absent. In this type of neurosyphilis there may be parenchymatous involvement or simply meningitic and vascular changes. It would seem, therefore, from these findings that the paretic curve in cerebrospinal syphilis would indicate parenchymatous involvement.

TABLE 4.—COLLOIDAL GOLD TESTS IN CEREBROSPINAL SYPHILIS

Author	Number of Cases	Paretic Curves	Syphilitic Curves	Chronic Type	Atypical Curves	Negative
Warwick and Nixon ¹⁵	26	3	10	—	9	4
Hammes ⁸	27	3	23	..	1	0
Black ¹⁶	39	1	15	20	—	3
Rawlings ⁷	59	1	33	..	14	3
Swalm and Mann ⁶	10	0	6	—	—	4
Miller, Brush, Hammes and Felton ⁴	15	4	9	—	—	2
Eskuchen ¹¹	13	1	12	—	—	—
Eicke ⁹	25	0	24	—	—	1
Cases in this series.....	31	15	6	—	6	4

15. Warwick and Nixon (Footnote 2).

16. Black, J. H.; Rosenberg, L., and McBride, R. B.: The Colloidal Gold Test, J. A. M. A. **69**:1855 (Dec. 1) 1917.

Tabes Dorsalis.—In tabes, the colloidal gold reaction is generally of the 0123321100³ type and either this reaction or a negative result was obtained in most of the cases. However, two cases gave a paretic curve with the first lumbar puncture fluid and before any treatment was given. One other case of tabes developed a paretic curve under treatment. These cases have been under observation for two years or more and have not shown any mental symptoms.

Undifferentiated Neurosyphilis.—The cases of undifferentiated neurosyphilis are those that show no clinical signs for differentiation and yet give a positive Wassermann reaction and other findings in the cerebrospinal fluid. A group of such cases has been described by Southard and Solomon¹⁷ under the term "paresis sine paresi." In this series of paretic curves appear the curves of seven patients with neurosyphilis who have been under observation for some time and yet have not shown any physical or mental symptoms that would warrant a diagnosis of either paresis or cerebrospinal syphilis.

17. Southard and Solomon: Latent Neurosyphilis and the Question of General Paresis Sine Paresi, Boston M. & S. J. **174**:8, 1915.

Multiple Sclerosis.—Recently, Moore¹⁸ has shown that a paretic curve is the usual reaction of the colloidal gold test in multiple sclerosis. He found that of twenty undoubted cases of multiple sclerosis, eighteen gave typical paretic curves. Among the cases of multiple sclerosis here there were five in which paretic curves were found. One of them has been confirmed by necropsy. This patient was admitted in 1913 with most of the characteristic symptoms of multiple sclerosis. She was later sent to another hospital, and there the same diagnosis was made. The patient died in January, 1920. At necropsy numerous areas of sclerosis were found in the cord and brain. These areas were distinct even macroscopically.

The spinal fluid findings were as follows: globulin, 2; albumin, 2; cells, 7; colloidal gold, 4444332000. The Wassermann reactions on the fluid and serum were twice negative.

TABLE 5.—CEREBROSPINAL FLUID FINDINGS IN FIVE CASES OF MULTIPLE SCLEROSIS

Case	Globulin	Albumin	Cells	Colloidal Gold Curve	Wassermann Reaction
5025	2	2	43	5555552210	Negative
6152	3	3	0	5555330000	Negative
F. H.	2	2	2	5555431000	Negative
	2	2	4	5555421000	Negative
B. C.	2	2	8	5555531000	Negative
	3	2	8	5555421000	Negative
	2	2	14	5555521100	Negative
2223	2	2	7	4444332000	Negative

The blood Wassermann reaction was negative several times in each case.

Brain Tumors.—Brain and cord tumors generally give what is called a "chronic curve," 0011232110.³ Many cases of brain tumor are to be found in state hospitals, but comparatively few such cases have been reported as giving paretic curves. Three cases with a diagnosis of brain tumor were found to have paretic readings in their spinal fluids, but only one of these could be definitely confirmed. This patient, whose spinal fluid gave a paretic curve on two different occasions, was found at operation to have a cerebellar tumor. The Wassermann reactions of the blood and spinal fluid were negative, and there was no evidence of syphilitic involvement of the central nervous system.

Larkin and Cornwall¹² report a case of psammona of the dura with a gold reading of 552221000. This is not a true brain tumor, and the reading is far removed from the typical paretic curve. Miller⁴ found a paretic curve in a case diagnosed as cerebral gumma. It is a question whether the new growth or the syphilis or a combination of both produced this reaction.

18. Moore: Cerebrospinal Fluid in Multiple Sclerosis, Arch. Int. Med. 25:58 (Jan.) 1920.

Epidemic Encephalitis.—The reports of the colloidal gold findings in the present epidemic of encephalitis are not numerous. Almost all the cases at this hospital and most of the cases reported in the literature have given a syphilitic type of curve (1123321000). Barker¹⁹ and his associates in their review of epidemic encephalitis report one case showing a "combined paretic and meningitic curve," and another case with an "atypical paretic curve." One case of the choreiform type of lethargic encephalitis at this hospital gave a typical paretic curve in the first fluid obtained after admission. Two tests were made with two different colloidal gold solutions and a paretic curve resulted with each.

CASE 14272.—A man, aged 26, whose family history and early development apparently were normal, in the first part of 1918, had an attack of rheumatism lasting about two months. About ten days before admission to the hospital (April 21, 1920) the attack of encephalitis started with headache and pains all over the body. These pains were especially marked in the arms and back. After a few days choreic movements began, and the patient soon became delirious. Several times the patient saw double. Previous to the choreic movements the patient slept most of the time and it was difficult to arouse him. He was admitted to the hospital in a semidelirious condition. The choreic movements were marked and quite typical. The temperature was 103 and the pulse rate 104. The white blood cells numbered 16,000.

Physical examination showed marked choreiform movements of the entire body. There was a faint systolic murmur at the mitral area. The deep reflexes were exaggerated, but no pathologic reflexes were present. The pupils were equal and regular and reacted equally well to light and accommodation. Otherwise the results of the physical and neurologic examinations were negative. Mentally, the patient remained in a semidelirious condition for a few days and then gradually the mental condition cleared up. The choreiform movements were checked by chloral. For about three weeks after the chloral was stopped, the patient was restless at night but would sleep almost all day, and it was difficult to arouse him. Fibrillary twitchings of the facial muscles were often noted. The temperature remained elevated for about a month. At the end of six weeks there was marked improvement and the patient had a fair insight into his condition.

The spinal fluid findings were: globulin, 2; albumin, 2; cells, 2 (small lymphocytes); colloidal gold, 5555542100. Wassermann reactions of the spinal fluid and the blood were negative on two occasions.

This and similar cases are soon to be reported in greater detail.

Alcoholic and Drug Psychoses.—Among the cases with paretic curves in the spinal fluid appears one case of Korsakoff's psychosis (alcoholic). The diagnosis was confirmed by another hospital, and the patient was later discharged as improved.

19. Barker, Cross and Erwin: On Epidemicacite and Subacute Nonsuppurative Inflammations of the Nervous System, Prevalent in the United States in 1918 and 1919, *Am. J. M. Sc.* **159**:157, 1920.

CASE 5909.—A colored man, aged 38, whose family history and early development were normal and who had had no venereal history, had always been somewhat alcoholic, but this tendency had markedly increased during the year previous to admission. He is said to have been mentally well until about four or five days before admission when he began to complain of headache and pain in his neck. After a visit from a friend, he immediately went to the telephone to call up this friend, forgetting that he had just been there. The following day he was quite confused.

On admission to the hospital (Dec. 6, 1915) he was disoriented for time and place. He was confused, and his answers were more or less irrelevant. When asked to count backward from 20 to 1, he had to give it up after trying for fifteen minutes and getting no further than 17. Memory for recent events was quite bad. He gave the ages of his three children as 2, 3 and 8 months, respectively. No distinct fabrication was noted during his stay.

Physical examination showed a weakness of the muscles of the lower back and of the hamstring muscles, so that there was great difficulty in standing. The knee reflexes were unequal, that of the right knee being diminished. The pupils were normal, and there was no sensory disturbance.

The patient was soon transferred to another hospital from which he was discharged six months later with a final diagnosis of Korsakoff's psychosis.

The spinal fluid findings were: globulin, 1; albumin, 1; cells 2 (small lymphocytes); colloidal gold curve, 5553210000; Wassermann reactions of the blood and spinal fluid were twice negative.

In the literature many instances of colloidal gold tests in alcoholic psychoses are given, and some show mild changes in the gold, but no paretic curves are recorded.

The cerebrospinal fluid from a patient with drug (heroin) psychosis at this hospital in 1914 gave a paretic curve. This was complicated by the presence of a doubtful Wassermann reaction in both the spinal fluid and the blood. However, the patient was sent to another hospital and remained there until September, 1919, when she was discharged with a final diagnosis of drug addiction (not insane).

Lowrey⁵ reports a case of drug addiction with a colloidal gold curve reading 4444100000, but there were positive Wassermann reactions in the serum and fluid. Moore¹⁸ states that a paretic curve is sometimes present in lead poisoning. No other gold reactions resembling a paretic curve could be found in many of the cases of drug addiction reported, although many did show mild changes in the syphilitic zone.

Undiagnosed Cases Definitely Not Paresis.—There were five other cases in which a definite diagnosis could not be made, but in which paresis could be ruled out.

CASE 4398.—The mother and several brothers of a white man, aged 40, died of tuberculosis. One sister was insane. The onset and course of the patient's illness was very indefinite. On admission (Feb. 21, 1915) he was found to be suffering from advanced pulmonary tuberculosis. There was involvement of the entire left lung and the upper and middle lobes of the

right lung, with cavity formation. The neurologic examination was negative. The patient slept most of the time. Temperature, pulse and respirations were increased. Memory was poor for recent and remote events. His grasp on his surroundings was poor. No hallucinations or delusions were found. The patient was unemotional and had no insight. He gradually grew worse and died eleven days after admission. No signs of meningeal involvement were found at any time and a necropsy examination was not obtained; therefore the diagnosis of tuberculous meningitis, which is the most probable one, could not be confirmed. A guinea-pig was inoculated, but owing to contamination no results were obtained.

The spinal fluid findings were: globulin, 3; albumin, 4; cells, 69 (small lymphocytes, 68 per cent., large lymphocytes, 22 per cent., plasma, 3 per cent., polymorphonuclears, 7 per cent.). The colloidal gold curve was 5555554321. The Wassermann reactions in the fluid and serum were twice negative.

Cases of tuberculous meningitis with parietic curves have been reported as shown in Table 6.

CASE 13364.—A white woman, aged 49, whose family history was negative, was healthy until 12 years old when she developed curvature of the spine; since then she has worn braces occasionally. The patient graduated from normal school when she was 21 and was considered a good student. She was somewhat of a "book worm." When about 28 years old she began to prepare to get married. She had a wedding dress made and even resigned her position, but there was no man in the case. The patient was at that time sent to a state hospital and remained there two months. She has not been normal since, according to her friends. She has a circle of friends whom she calls her "arch friends" and who have a "program" of persecution. After making several attempts to kill one of them she was sent to this hospital for observation (Oct. 14, 1919).

On admission her mental condition showed nothing abnormal except a wealth of paranoid ideas that she had maintained for twenty years. Among her "arch friends" was a man or male spirit whom she prepared to marry twenty years ago. Since then this spirit has had sexual intercourse with her by means of electricity. The patient had a religion all her own in which her hands play an important part. Auditory and visual hallucinations were denied. She had some insight at times. Personality was well retained.

Physical examination revealed a right upper thoracic scoliosis and a resulting deformity of the chest. There was a scar on her right breast where the entire right mammary gland was removed a few weeks before entering this hospital. There was a lump the size of a walnut in the left breast and palpable axillary glands. The neurologic examination was negative.

After four months the patient was transferred to another hospital from which she was later discharged as improved. The diagnosis remained "paranoid condition."

The spinal fluid findings were: globulin, 1; albumin, negative (?); cells, 3 small lymphocytes, 1 large lymphocyte; colloidal gold curve, 5555531000 (two tests). The Wassermann reactions on the spinal fluid and serum were twice negative.

CASE 13179.—A white man, aged 26 (?), whose family history was somewhat defective, was a full term, healthy baby but could not talk until he was 4 years old. He had smoked excessively since he was 8 years old. He was quarrelsome and mischievous as a child, but quite honest. He was backward

in school and did not learn to read very well. He repeated the fourth and fifth grades and never got much further. At 13 he ran away from school. In 1914 he was arrested for stealing and sent to this hospital. Because of an apparent character change and abnormal emotional reaction the condition was diagnosed as dementia praecox simplex, and the patient was transferred to another hospital. After three months there he was discharged and went back to work as a machinist. His mental age at that time was 11.1. In 1917 he enlisted and was sent to France. He was returned to this country in January, 1919, after being gassed twice, and was placed at various base hospitals. Finally, he remained away without official leave for eleven days and was sentenced to serve three years. After serving four months he was again sent to this hospital for observation.

On admission (Sept. 8, 1919) he was correctly oriented. Memory was somewhat bad and the details of his story did not check up well. Calculation ability and school knowledge were rather poor. No evidence of delusions was found, but he admitted hearing bells and voices while in France. Emotional responses were quite normal, and personality was well retained. There was no disturbance of thought processes. His mental age was not tested again. Physically, he was well developed and well nourished. His breath was foul, and the tongue was tremulous, rough and fissured. His hands and feet were cold and cyanotic. The pupils were irregular and reacted sluggishly to light and accommodation through a narrow arc. The knee reflexes were sluggish but equal. Sensation was normal. There was no Romberg sign.

The spinal fluid findings were: globulin, 1; albumin, 1; cells, 41 small lymphocytes; colloidal gold curve, 5554432100. The colloidal gold test was repeated with a different colloidal gold solution, and practically the same result was obtained. The Wassermann reactions on the serum and spinal fluid were consistently negative several times. The patient was given provocative arspenamin, but the Wassermann test remained negative. After the provocative treatment another lumbar puncture was made with the following results: globulin, 1; albumin, 1; cells, 20 small lymphocytes; colloidal gold curve, 0123220000.

Since being discharged the patient has gone back into the transport service and is getting along well. The blood Wassermann test remains negative.

CASE 13430.—The patient gave a history of excessive alcoholism for several years before admission to the hospital with an increase in the amount of alcohol consumed just prior to entrance. A history of a chancre about a year and a half before admission complicated the case. The patient became hallucinated about six months before he was sent to the hospital. On entrance he had marked hallucinations and delusions. The hallucinations were auditory and were mostly in the third person. He thought that he was persecuted by the Catholics and that they had electromagnets which controlled his thoughts. There were numerous other delusions of persecution. There was no obvious disintegration of personality and no memory loss. The patient was superior in the psychometric test. Orientation was intact, and there was no disorder of consciousness. Emotional tone was in keeping with his delusions. The physical and neurologic examinations were entirely negative. After six weeks in the hospital the hallucinations had almost completely disappeared, and the patient had good insight.

The spinal fluid findings in the first fluid tested were: globulin, 1; albumin, 1; cells, 8 small lymphocytes; colloidal gold curve, 555321000. The Wassermann test was negative. The patient was given several intravenous injections

of arsphenamin, but the blood and spinal fluid Wassermann reactions remained negative, while the other spinal fluid tests were practically the same on two subsequent examinations.

CASE 5364.—A girl, aged 23, admitted to this hospital in 1915, was of the defective delinquent type, and this was the provisional diagnosis, although her case was later left unclassified. She was moody, depressed and easily irritated. She had delusions of persecution, but no hallucinations. Physical examination revealed only poorly reacting pupils. The diagnosis of manic depressive insanity was considered, and it is possible that it was a case of neurosyphilis although the Wassermann reaction was negative. The patient was sent to another hospital from which she was later discharged as improved.

The spinal fluid findings were: globulin, 2; albumin, 2; cells, 13; colloidal gold curve, 5555543310. The Wassermann test was negative.

TABLE 6.—MISCELLANEOUS CASES; NEUROSYPHILIS AND MULTIPLE SCLEROSIS EXCLUDED

Author	Number of Cases	Source or Type of Cases	Paretic Curves	Conditions
Warwick and Nixon ²⁰ ..	245	General hospital.....	1	Brain abscess
Lowrey ⁹	115	State hospital.....	1 (?)	Drug addict
Larkin and Cornwall ²¹	114	Epilepsy.....	1	Epilepsy
Stevens ²²	38	Mongolian idiocy.....	0	
Hammes ⁸	92	General hospital.....	0	
Jeans and Johnson ²³ ..	100	Poliomyelitis.....	0	
Weston ¹⁴	165	State hospital.....	2	Undiagnosed
Vogel ²⁴	52	General hospital.....	1	Questionable
Rawlings ⁷	319	Psychopathic hospital	1	Defective
Williams and Burdick ²⁵	79	0	
Swalm and Mann ⁶	27	0	
Miller, Brush, Hammes and Felton ⁴	102	Psychopathic hospital	2	Brain abscess; tuberculous meningitis
Eskuchen ¹¹	56	0	
Jaeger and Goldstein ¹⁰	54	3	Brain abscess; tuberculous meningitis; epilepsy
Elcke ⁹	222	0	
Larkin and Cornwall ¹²	115	4	Eclampsia, 2; psammoma of Cerebellar tumor, 1; Korsakoff's psychosis, 1; encephalitis, 1; drug addict, 1; tuberculous meningitis, (?) 1; undiagnosed, 4
Present series.....	6,500	Psychopathic hospital	9	

20. Warwick and Nixon (Footnote 2).

21. Larkin and Cornwall: Spinal Fluid in Epilepsy, J. Lab. & Clin. Med. **4**:352, 1919.

22. Stevens, H. C.: The Spinal Fluid in Mongolian Idiocy, J. A. M. A. **66**:1373 (April 29) 1916.

23. Jeans and Johnston: Am. J. Dis. Child. **13**:329, 1917.

24. Vogel, K. M.: The Nature and Interpretation of the Colloidal Gold Reaction, Arch. Int. Med. **22**:496 (Oct.) 1918.

25. Williams and Burdick: Lange Colloidal Gold Reaction, Colorado Medicine **13**:103, 1915.

Rawlings ⁷ reports a case with a paretic curve in which the diagnosis was "defective."

In the literature from other similar hospitals, from general hospitals and from army hospitals, other conditions giving rise to paretic curves were found. These, together with the cases from this hospital, are given in Table 6, which gives the miscellaneous cases reported,

excluding neurosyphilis and multiple sclerosis, because paretic curves are to be expected in such cases.

VARIOUS OTHER CONSIDERATIONS

Relation to Other Tests.—The nonconcomitance of spinal fluid tests has been fully discussed by Solomon.²⁶ He concludes that any one of the usual five tests may be present or absent when the others are present, because each reaction is produced by a distinct chemical element that may be present alone. The relation to the Wassermann test is shown in Table 3 and has been discussed by Hammes.⁸

Effects of Treatment.—The colloidal gold reaction may vary considerably under treatment just as the other spinal fluid and blood tests do. With intensive treatment in favorable cases the tendency is for the paretic curve to become irregular or change to the "syphilitic type" and then finally to become negative. We have many such instances of which the following is a good example:

TABLE 7.—VARIATION OF COLLOIDAL GOLD REACTION UNDER TREATMENT *

Date	Colloidal Gold Curve	Globulin	Albumin	Cells	Wassermann Reaction	
					Cerebrospinal Fluid	Serum
3/21/15	5555554321	4	4	80	Positive	Positive
3/31/15	5555553100	4	4	96	Positive	Doubtful
4/ 7/15	5555543200	3	3	67	Positive	Doubtful
5/ 8/15	0011100000	2	1	8	Doubtful	Doubtful
8/13/15	1000000000	1	1	5	Negative	Negative
10/21/15	0000000000	0	Normal	4	Negative	Negative

* Intensive intraspinal and intravenous treatment.

The time of disappearance of the paretic curve is variable as has been pointed out by Solomon.²⁶ According to Neyman and Frush,²⁷ the paretic curve is the last of the positive spinal fluid reactions, with the exception of the globulin test, to disappear under treatment.

Improvement in the paretic curve is not the rule in treated cases. In fact, the reaction may become more pronounced either temporarily or permanently, just as the Wassermann reaction has been observed to do. The changes in the colloidal gold test may not parallel the clinical change in a treated patient. At present the paretic curve, as well as the other reactions of a patient who is having an almost perfect clinical remission, remain strongly positive in spite of intensive treatment. The opposite condition has also been found in a few instances. However, when there is clinical improvement and the other tests tend

26. Solomon, H. C.: Nonconcomitance of Spinal Fluid Tests, *Arch. Neurol. & Psychiat.* **3**:49 (Jan.) 1920.

27. Neymann and Brush: Treatment of General Paralysis, *Arch. Int. Med.* **22**:245, 1918.

to clear up, the gold reaction will, in the great majority of cases, follow the same course.

Provocative Colloidal Gold Test.—McBride¹⁶ made the statement that there is no colloidal gold reaction to provocative treatment but, on the other hand, Warwick and Nixon²⁰ and others speak of finding a provocative colloidal gold reaction. We have several cases in which the colloidal gold reaction was at first negative, but after a few treatments, a typical paretic curve appeared and remained for some time. This has happened too often to be merely coincidental. The cases in Table 8 are illustrative of this, and it will be noted that the other findings may also become more marked.

TABLE 8.—COLLOIDAL GOLD REACTIONS TO PROVOCATIVE TREATMENT

Date	Colloidal Gold Curve	Globulin	Albumin	Cells	Wassermann Reactions		Treatment
					Cerebrospinal Fluid	Serum	
Case 13013 1/24/19 0011230000	... ?	... ? 139 Negative Positive	Between dates given, 33 intravenous injections and 7 grains of mercury; no previous treatment
10/ 4/19	5444310000	1	1	50	555000	Negative	
Case C. A. 1/15/19 0000000000	... 0	... N 2 Positive Positive	No previous treatment; between dates given, 19 intravenous injections and 11 grains of mercury
1/20/20	5555531000	1	2	2	555500	Positive	
Case W. F. 11/18/15	1233330000	3	3	20	55520	Negative	3.3 gm. arsphenamin 2.2 gm. arsphenamin 1.8 gm. arsphenamin
12/15/15	5555553100	3	2	12	50420	Doubtful	
2/24/16	3333220000	2	2	2	Doubtful	Negative	
1/26/17	0000000000	1	1	2	53111	Negative	
Case W. Me. 6/14/19 0000001010	... 0	... N 0 Negative Negative	No previous treatment 1.6 gm. arsphenamin 1.3 gm. arsphenamin
8/ 2/19	5555431000	1	1	13	Negative	Negative	
9/ 6/19	5444321000	1	1	7	Negative	Negative	
10/11/19	5433211000	1	1	1	Negative	Negative	
Case E. F. 8/10/18	1112321000 1114321000	2	2	116	500000	Positive	0.8 gm. arsphenamin and 1 intraspinal injection; 4 gm. arsphenamin
12/ 2/18	5554443221	1	1	2	555540	Positive	
11/ 2/19	5554443210	1	1	3	555550	Negative	
Case F. M. 5/15/18	0001332110	2	1	56	Doubtful	8.4 gm. arsphenamin 2.3 gm. arsphenamin 2.6 gm. arsphenamin
3/15/19	1222333310	0	N	2	5000000	Negative	
8/ 9/19	5433310000	0	1	7	2200000	Negative	
1/24/20	0111110000	1	1	5	Negative	Negative	

It sometimes happens that without the usual treatment a paretic curve may appear after three or four lumbar punctures. This may in some cases be coincidental with the development of the disease, or it may be that the repeated lumbar punctures act as a provocation.

Effect of Contamination.—Various contaminations of the cerebrospinal fluid may modify the paretic curve. The fluid may become contaminated by using dirty test tubes or old corks that have been used with drugs; by the growth of yeasts or molds; by blood obtained when the lumbar puncture is made; and by various other foreign substances. In general, such contaminations may change a syphilitic type of fluid into a paretic type, but will seldom if ever produce a paretic curve in an absolutely normal fluid. A paretic fluid that has become contaminated will usually give a somewhat irregular reading resembling a paretic curve and may become practically negative.

With due care all these sources of contamination may be avoided with the exception of blood, which will sometimes be obtained in spite of all precautions. After repeated experiments of adding various quantities of blood to known normal fluids, it was found impossible to produce a reading that could be called a paretic curve. Such fluids would show marked and irregular color changes in the middle or at the end of higher dilutions. Addition of blood to the syphilitic type of fluid would occasionally give a paretic curve. Blood added to a known paretic fluid would generally produce an irregular reading but not a negative one. However, in cases contaminated at the time of puncture, paretic curves have been noted in normal cases and vice versa.

The effects of the presence of blood in the cerebrospinal fluid has been discussed by Black¹⁶ and Kellert.²⁸

CONCLUSIONS

1. The paretic gold curve does not always indicate paresis, but, if used in conjunction with the other spinal fluid tests, it is a most valuable test for this disease. In some instances, this reaction is the deciding factor in the diagnosis of general paralysis.

2. The vast majority of cases of paresis will give a paretic type of gold reaction. In a case that does not give such a reaction after more than one examination, the diagnosis of paresis is always questionable.

3. The presence of paretic curves in almost 50 per cent. of the cases of cerebrospinal syphilis shows that a paretic curve does not serve to differentiate paresis from this condition. However, the absence of a typical reading points strongly to cerebrospinal syphilis when the differential diagnosis lies between these two conditions.

4. A paretic or marked syphilitic curve is to be expected in multiple sclerosis.

28. Kellert: Observations on Colloidal Gold Reactions with Cerebrospinal Fluid, *Am. J. M. Sc.* **159**:257, 1920.

5. Besides paresis and multiple sclerosis, a paretic curve is occasionally found in the following conditions: tabes dorsalis, cerebrospinal syphilis, undifferentiated neurosyphilis, brain tumor, brain abscess, encephalitis, tuberculous meningitis, Korsakoff's psychosis (alcoholic), eclampsia, epilepsy, lead poisoning (?) and drug addiction (?).

6. The pathology of the above conditions would indicate more or less destruction of nerve cells in the brain, or, in other words, a parenchymatous involvement. It may be concluded, therefore, that a paretic curve points toward parenchymatous involvement of the brain, while the milder gold curves are obtained in meningitis, vascular disease and other conditions. The paretic curve is not only of diagnostic value, but also of prognostic value.

DEGENERATION GRANULES IN BRAIN CELLS IN EPIDEMIC ENCEPHALITIS*

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In the routine examination of sections from the brain of a case of epidemic encephalitis, minute granules were found in degenerated nerve cells in the areas involved by the disease process. Although similar granules in many diseases of the central nervous system have been described in German publications, notably by Alzheimer¹ and Achucarro,² to my knowledge their presence in cases of epidemic encephalitis has not previously been noted, and it therefore seems worth while to describe them as part of the microscopic pathology of this disease.

The granules are best demonstrated in tissues fixed in Zenker's or Helly's fluid, embedded in paraffin and stained with eosin-methylene blue or by Bensley's modification of the Altmann stain (acid-fuchsin-methyl-green). To bring them out with eosin we used a combination of equal parts of strong alcoholic and 5 per cent. aqueous eosin, staining forty minutes at 56 C. This stain, made up by mistake for routine eosin-methylene blue staining, was the one with which we first demonstrated the granules. With it the red blood cells in the sections appeared a deep copper red, the stroma bright pink, and the granules bright red or purple red. With Bensley's stain they appeared a somewhat brighter red.

As demonstrated by these stains, the granules appear as minute spherical masses, measuring from about 0.5 to 2 microns in diameter, sharply outlined and highly refractile. The largest ones are nearly half as large as the nucleoli found in these nerve cells, but they are not so large as even the smallest Negri bodies of rabies, which stain somewhat similarly with acid fuchsin. The larger ones often have a minute colorless center, as though the stain had not thoroughly penetrated. They also are frequently surrounded by a clear zone in the cell cytoplasm. In number they vary greatly, and their number

* From the Medical Pathological Laboratory of the Presbyterian Hospital, Department of Pathology, College of Physicians and Surgeons, Columbia University.

1. Alzheimer, A.: *Histologische und Histopathologische Arbeiten* **3**:491, 1910.

2. Achucarro, N.: *Histologische und Histopathologische Arbeiten* **3**:174, 1910.

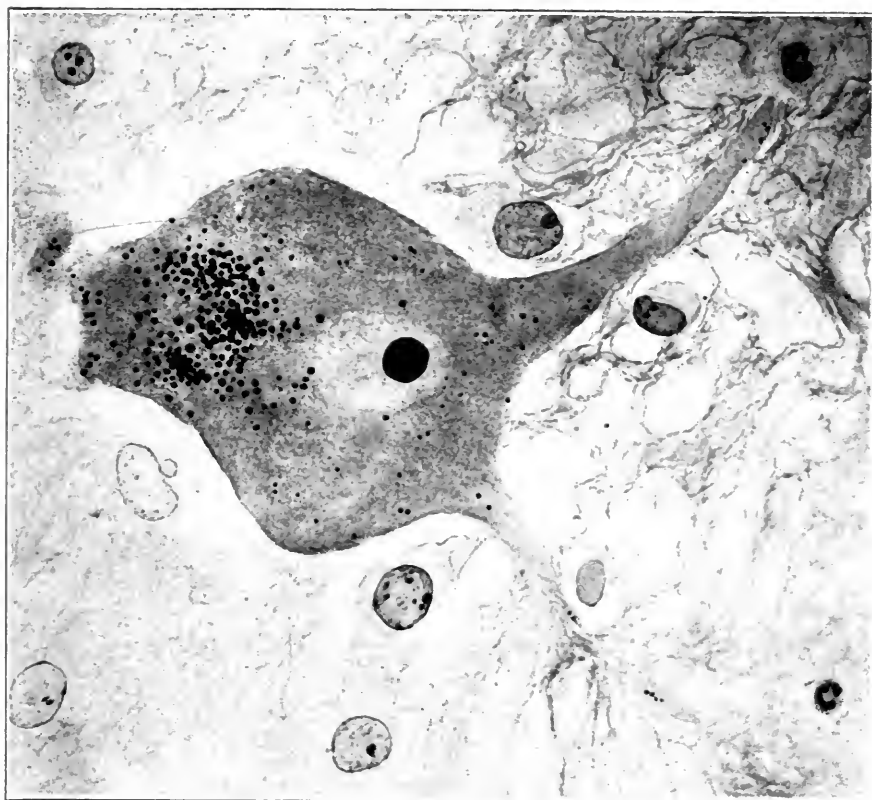


Fig. 1.—Degenerated ganglion cell from a basal ganglion of a case of epidemic encephalitis, showing degeneration granules in the cytoplasm.



Fig. 2.—Smaller ganglion cells from the same section, showing degeneration granules and beginning neuronophagia.

appears to have no relation to the degree of degeneration of the nerve cell in which they occur. Frequently only a single granule is found in a cell, while other cells, whose appearance is no more abnormal, contain forty or fifty granules. Some definitely degenerated cells contain no granules in the section studied, though parts of the cell not cut may have contained them. The nerve cells in which these granules are found are always more or less abnormal in appearance, as evidenced by loss of definite outline, dissolution of Nissl substance, acidophilic reaction of cytoplasm—particularly in the region of the granules—accumulation of lipochrome pigment in the cytoplasm, swelling or fragmentation of the nucleus, or beginning phagocytosis of the nerve cell by invading wandering cells. The degenerated cells are found only in areas in which there are other lesions characteristic of the disease, namely, perivascular hemorrhage, or perivascular or diffuse lymphocytic infiltration. In the cases of epidemic encephalitis studied, granules have been found in the occipital cortex, the basal ganglions,

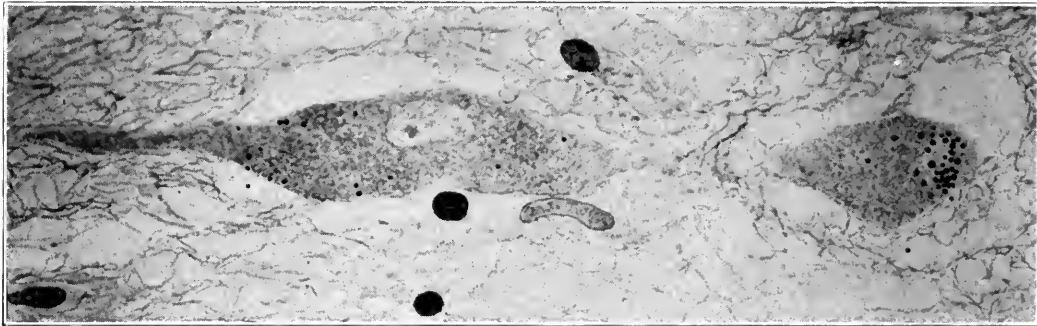


Fig. 3.—Ganglion cells from the basal ganglions of another case of epidemic encephalitis, showing degeneration granules, some of which are outside the cell due to partial lysis of the cytoplasm.

the pons and the medulla. They have not been found in the anterior horn cells of the spinal cord, although one case showed marked perivascular lymphocyte infiltration in the spinal cord, and definite degeneration of anterior horn cells. In the nerve cells of the posterior root ganglions of the same case granules were found which gave the same staining reaction as the granules I am describing, but they were more irregular in outline, were more numerous and were usually arranged in small rosettes.

No extensive study has been made of the chemical nature of the granules. Sections treated with 10 or 20 per cent. nitric acid for five minutes before staining show the granules as brilliantly as before, whereas sections similarly treated with 5 per cent. sodium hydroxid show them hazily outlined and dull red in color. Sections fixed in

liquor formaldehydi show the granules poorly or not at all, the acid-fuchsin stain bringing them out better than eosin.

That the granules are not specific to epidemic encephalitis is shown by the fact that they have been demonstrated in the basal ganglions, pons and medulla, and once in the spinal cord in four cases of poliomyelitis, and in the basal ganglions in a case of tuberculous meningitis associated with lymphocytic infiltration of the brain stem. Granules similar to those found in the dorsal root ganglions of the case of epidemic encephalitis mentioned were also found in the dorsal root ganglion cells of a case of *tabes dorsalis*. On the other hand, several cases coming to necropsy, which showed no clinical or anatomic involvement of the central nervous system, have failed to show these granules in the nerve cells of the brain.

Without more extended study it is perhaps unwise to discuss the possible derivation of these bodies. Their constant association with degenerative changes in the nerve cells indicates that they are some form of cytoplasmic degeneration. The most obvious source, because they stain with mitochondrial stains, would be the mitochondria themselves. The studies of McCann³ on the posterior root ganglions of poliomyelitic monkeys have shown that the mitochondrial filaments may persist in the presence of pathologic changes in the ganglion cells. The granules described in this paper, if derived from mitochondria, are necessarily much altered in their chemical structure, as they are resistant to postmortem autolysis and to the action of strong acid and alkali, and are demonstrable by staining methods which do not bring out mitochondria in the same cells.

3. McCann, G. F.: *J. Exper. Med.* **27**:31, 1918.

THE PSYCHIATRIC FEATURES OF SO-CALLED LETHARGIC ENCEPHALITIS

CASE REPORTS AND A REVIEW OF THE LITERATURE

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Probably no single topic in the field of nervous and mental diseases has, within the past year, received more attention than so-called lethargic encephalitis, and yet the vast literature on the subject contains little concerning the psychiatric aspect of this disorder besides that given in a few papers noted later under a discussion of the literature.

We came in direct or indirect contact with about fifty or more cases of this disorder during the winter and spring of 1920. With the exception of the four cases reported in this paper, especial attention was not given to the mental picture except in regard to the condition called "substupor" that was so pronounced in most of the cases. The physicians who had charge of most of the patients were more particularly interested in cranial nerve palsies, lost knee jerks, spasmodic muscular twitchings, bladder disturbances, nystagmus and a host of other neuropathologic findings. We present this subject because we studied the mental conditions of our patients and because three, if not all, of our cases occurred in persons who had, before their infection, exhibited more or less pronounced neurotic tendencies. One had been under observation for some time to determine whether he was suffering from so-called petit mal epilepsy or from anxiety attacks of hysteria. Another had an antecedent history of obsessions; a third was a highly unstable and emotional child, and the fourth was of distinctly high strung temperament. This raises the question, perhaps of a will-o'-the-wisp character, as to whether the neurotic tendencies predisposed the patient to an attack of encephalitis or whether the attack threw into bold relief the psychiatric picture in the case.

REPORT OF CASES

CASE 1.—*History*.—B. B., a white boy, aged 9; whose maternal grandmother died at the age of 53 of a "nervous skin eruption" and whose paternal grandfather died of apoplexy at the age of 69, had been a patient at Pontiac State Hospital for some time. His mother, aged 29, was emotionally unstable. The father was apparently normal, a successful salesman. He was suing the

patient's mother for divorce on the ground that her youngest child (10 weeks old at the time the patient was first seen) was not his. The mother was bringing counter charges of infidelity.

Gestation and birth were negative. He had had measles, mumps and whooping cough after beginning school. During the past three or four years he had always become fatigued easily on exertion, and on several occasions had had episodes of sleeping for two or three days following exertion.

Make-Up of the Patient.—He was easily controlled and of a happy disposition, with the exception of frequent spells of emotional instability not unlike those of the mother. He was fond of boyish sports. His mentality was above the average; this was confirmed by intellectual tests.

Present Illness.—He contracted what was diagnosed by the family physician as influenza in February, 1920. His temperature was elevated for eleven days. Recovery was good except for "lameness" in the right leg and knee. This condition was intermittent. He had been out of school twenty-three half days. He returned to school about March 8, doing well until April 14, 1920, when his teacher noticed that he was slow in arithmetic, and that his handwriting was tremulous to the point of being illegible. At this time the mother noticed that he continually tossed about in bed with movements not unlike those of acute chorea. Intention tremor was noticed especially when he tried to eat or drink. He had to be fed for this reason. He was unable to button his clothes. His speech was thick. He had progressive emotional instability.

Examination.—The patient was first seen April 19, 1920. His face was flushed. The temperature was 100.5 F.; pulse rate, 108. Blood pressure was systolic 95, diastolic 58. The heart beat was irregular and pounding, but no murmurs were detected. Neurologic examination showed wide choreiform movements of all four extremities. The throat was difficult to examine because the head was in constant motion. Knee reflexes were not obtained. The chief mental symptom was emotional instability.

Clinical Course.—April 20, lumbar puncture showed a clear fluid, twenty-one cells, negative globulin and a flat gold curve. There was moderate leukocytosis and 13,500 white cells were found. Blood culture was negative. There was a cell count of twenty-three, and of nineteen on the occasion of two later spinal punctures made within the following week. After that the spinal fluid was negative on two occasions and within two weeks the blood count was normal, and all neurologic symptoms had disappeared, except choreiform movements of the hands, and the absence of knee reflexes. These in turn disappeared within a month after the patient was first seen.

Mental Picture.—The outstanding feature, both before and after the patient came under the writers' observation, was marked emotional instability. There never was a period or stage that could be designated as the lethargic or stuporous stage. On the third day after he was examined there was a short period of unclearness or mental confusion. After one of his evening meals he appeared half delirious, getting out of bed, and then trying to get in again. He appeared to realize that he had defecated in the bed, but seemed to have lost the power of speech, only pointing to the soiled linen. When power of speech returned there seemed to be a transient aphasia, or paraphasia. For this episode he later had no memory. He was discharged as recovered, six weeks after he was first seen. At present he shows no evidence of his illness except a slight emotional instability.

Had it not been for the spinal fluid findings, the leukocytosis, and the temporary loss of the patellar reflexes, the case would have been regarded as one of Sydenham's chorea.

The outstanding mental symptoms were: emotional instability, inability to perform simple arithmetical calculations, and a brief period of unclearness with amnesia for the same on recovery. There was no evidence of mental deterioration or defect as a result of the illness. It should not be forgotten that the patient was emotionally unstable during a state of physical health, and that there was a taint in the family history.

CASE 2.—History.—A. M., a young man aged 28, single, white, a machinist, had a negative family history. The previous medical history was unimportant.

Make-Up of the Patient.—He was somewhat high strung. He always worked as though under tension, frequently late into the night with his books, even after long hours in the factory as an automobile mechanic. He frequently talked of inventions and machinery. He was fairly sociable in his habits; somewhat sentimental.

Present Illness.—On Tuesday, April 13, 1920, the patient began to be troubled with terrifying dreams, such as concerning a bear getting under his bed, and creeping up the bed after him. He complained of general malaise, but felt the necessity of keeping at his work. He obtained little or no sleep for the next four nights. He had anorexia, and was troubled with nausea and vomiting on the way to, or at, work.

Absentmindedness was noticed by his landlady. He would start to work without his door-key, papers, pencils, etc. On Tuesday, April 15, he started to work in the rain without his umbrella. He returned to the back stairway for his rubbers when they were in the front hallway. Choreiform movements began that night. He had subjective sensation of teeth falling out, relieved only by biting the hot-water bottle.

For the week preceding these symptoms the patient had been troubled with sore throat. Friday, April 16, frequent yawning and stretching movements, difficulty in breathing and ambling gait were noticed. That night generalized thrashing or tossing movements of entire body were present. The family physician was called. The movements were not affected by morphin, $\frac{3}{4}$ grain, or veronal, 30 grains, in twenty-four hours.

Examination.—The patient was first seen on Sunday, April 18, at noon. His face was flushed, and he was tossing violently about in bed, his movements being not unlike those of one having a severe hysterical attack. He would throw himself in the air, the rebound almost severing the springs from the bed. He answered questions, however, and said he suffered no pain. He kept his eyes closed most of the time. He could be quieted by laying a hand on his trunk or extremities.

Neurologic examination disclosed nothing unusual. Pupillary responses, and extra-ocular movements were normal. There was no nystagmus. Knee reflexes were normal, as were the other deep tendon reflexes. The superficial reflexes were unimpaired. Blood pressure was normal. The temperature was 99 F. He was admitted to Harper Hospital on April 18, 1920. For the next two days thrashing movements continued, at times almost becoming opisthotonos. At all times he responded rationally to questions.

On the third day the knee reflexes were questionably absent and the abdominal reflexes were not obtained. There was a bilateral weakness of the sixth nerve, more pronounced on the left. Morphin had no effect on the spasmodic

movements. Apomorphin did not cause vomiting, but did have a quieting effect. The laboratory findings were:

1. Spinal fluid: negative Wassermann reaction; cell count, 140; decided increase in pressure; crystal clear fluid; globulin, positive; mastoid and gold curves negative.

—2. Blood: negative Wassermann reaction; blood nitrogen, 29 mg. per 100 c.c.; blood count: red, cells, 4,650,000; white cells, 9,000; differential count, negative; red cells, normal in size and staining reaction; hemoglobin, 80 per cent.

3. Urine: negative.

On the first day of admission to the hospital, the temperature rose to 102 F., and gradually fell to normal by the fourth day.

Mental Picture.—He had terrifying dreams and was unable to concentrate. He responded well to questions. On the day of his admission to the hospital, insight was poor, the patient regarding himself as well. As proof he stated that he was no longer troubled with certain numbers running constantly through his mind, such as "16 Z 1185," a number on a machine part which he had been making, or "167753," the engine number of his automobile. "If I had nothing to read, I would keep repeating these numbers. I was sick then; I am well now." On April 20, the patient stated that he had a total amnesia for a period of four days from April 16 to 19 inclusive, and he did not remember his terrifying dreams, the visits of his physicians, or his admission to the hospital. During the period of his amnesia he frequently manipulated his genitals, even when the nurse was in the room. Of this he also had no memory, but he thought he had had a diplopia for a few minutes on the night of the 16th when he was first taken acutely ill.

Clinical Course.—After the fourth day in the hospital, improvement, both mentally and physically, was rapid. For a week there was a period of mild drowsiness from which he could be easily aroused. He was discharged one month after admission. At that time the chief residual of his illness was a slight blurring of vision. He could not read for over twenty minutes without fatigue. There was still a noticeable weakness of the external recti and a slight arrhythmia of the heart. Three months after his discharge from the hospital, he was able to return to work, and the only residual of his previous illness was a drowsiness which usually appeared about 9 o'clock in the morning while he was at work. "Even a few seconds of dozing" would make him "good for the rest of the day."

The principal mental symptoms in this case are:

1. Terrifying dreams.
2. Absent-mindedness and diminished power of concentration.
3. Delirium of a mild character with complete amnesia for the period of delirium. This period was marked by spasmodic twitchings and generalized movements, manipulation of the genital organs, mild degree of euphoria and lack of insight.
4. Short periods of substupor immediately following the irritative stage. This substuporous condition was not marked. He answered questions readily, but usually dropped off into a half drowsy state after the questions were answered. This period continued for about a week. At no time was it difficult to get his attention.
5. Three months after his discharge from the hospital there were brief moments of this drowsy tendency, occurring usually in the morning.

CASE 3.—*History*.¹—S. O., a single man aged 23, a Russian Jew, had a negative family history, except for the fact that he and many of his brothers and sisters, nine in number, suffered from enuresis, several as late as the sixteenth year. He came to this country from Russia at the age of 15. He had received a high school education. For several years he had assisted an older brother in the mercantile business, and for three years he had managed a small store himself.

About 1915, the patient began to have attacks, or "spells," of an epileptiform or hysteroid character. They would usually come on in the morning just after he had arisen, and would last not longer than a minute. There were aura, particularly peculiar sensations and paresthesias in the right arm. He never fell, but was able to get to a chair or bed. He would then become tense and rigid and appeared to lose consciousness for a short interval. He was admitted to the State Psychopathic Hospital at Ann Arbor, for observation, on Nov. 19, 1917, and was discharged December 11 of the same year.

Physical examination was practically negative. Neurologic examination showed a myopic fundus with a congenital choroidal lesion in the left eye. There was questionable horizontal nystagmus. There was a slight facial asymmetry. During the examination, muscles of the thighs and calves showed twitching and both limbs exhibited tremors. Patellar and Achilles' reflexes were active.

Laboratory findings, including the blood Wassermann reaction, were negative.

During his residence at Ann Arbor the patient had about four mild convulsive seizures. All occurred during the night and left no bad after-effects. On Dec. 8, 1917, he had several seizures of mild character with momentary confusion, accompanied by slight general muscular twitchings. His case was diagnosed as epilepsy, nocturnal petit mal attacks.

Make-Up of the Patient.—He was quick-tempered, but sociable and popular with his associates. For two years his habits were somewhat irregular and a source of concern to his family. He denied venereal infection.

Present Illness.—On Dec. 31, 1919, the writer was called in consultation. It was learned that his so-called petit mal attacks had gradually lessened in frequency after his discharge from Ann Arbor, and that for three months prior to December 31, these attacks had disappeared entirely. Even before that time an attack could be aborted by awakening him during the night or by simply turning on the electric lights in his room.

On or about Dec. 17, 1919, the patient had caught cold following the opening of some new stores. He appeared to recover, but had a slight relapse, and was confined to bed a day or two about December 22. On Christmas day he began to suffer what was described as "neuritis" affecting one part of the body, then another, but never the joints. He was sent to Mount Clemens for the baths, but while there he grew steadily worse, the chief symptom being severe spasmodic twitchings of the muscles of the trunk, which the brother thought might be "hysterical."

On Jan. 21, 1920, he was admitted to Harper Hospital in the Neurological Service.

On the day of admission the neurologic examination was negative, except for irregular jerking movements of the abdominal muscles.

1. History of this case from Nov. 19, 1917, to Dec. 11, 1917, was obtained by courtesy of Dr. Albert M. Barrett, Director of State Psychopathic Hospital, Ann Arbor, Mich. S. P. H. No. 2191.

Mentally he appeared to be in a dream state or a mild delirium. He constantly kept his eyes closed and emitted grunting noises not unlike those of an animal. He was restless, and continually picked at the bed clothes; at other times he would cover his head with the bedspread not unlike a manic or praecox patient. He would frequently ask for the urinal, get out of bed in a half-dazed way, stand as if puzzled, and would not urinate. When asked where he was suffering pain, he would answer, "Six dollars." Three tenths of a grain of apomorphin was given with no apparent effect.

On January 2, he was disoriented for time, person and place. One physician was a "policeman," and the other a "mail collector."

The neurologic examination was still negative on January 2, but lumbar puncture gave a positive globulin test and 90 cells in the fluid. Other important laboratory findings, January 7, were:

1. Urine, negative.
2. Blood count: red cells, 4,900,000; white cells, 14,800.
3. Blood Wassermann, blood culture and blood nitrogen, all negative.
4. Spinal fluid Wassermann and culture negative.
5. Lange and Mastic curves negative.

From that time on the patient's symptoms became more grave. His temperature began to rise, reaching as high as 104 F. The spasmodic contractions of the abdominal muscles continued; the right pupil became sluggish; the tendon reflexes, especially the patellars were somewhat delayed; there was a noticeable intention tremor, and speech became somewhat thick. Cardiovascular and respiratory symptoms appeared, and it became necessary by January 10 to inject normal salt solution intravenously. For several days before this the "substuporous" mental state appeared, or perhaps what might better still be called a "low grade stupor," from which the patient could always be aroused sufficiently to answer questions, though his mentality was as a rule unclear. This stupor was more marked in the morning.

On January 5, the patient's spinal fluid agglutinated the blood of five or six other similar cases then in the hospital, and this was regarded as a favorable symptom. Nevertheless he steadily grew worse. His condition became so bad that two blood transfusions were done, the donors being his brothers. In spite of this he died in a state of coma on January 26, twenty-six days after admission. Unfortunately, necropsy was refused by the family.

It should be added that during the last two weeks of his illness bladder symptoms appeared, the urine being literally ejaculated frequently the full length of the bed.

Mental Picture.—The principal mental features in this case are:

1. Several siblings with enuresis up to as late as the sixteenth year.
2. The patient was subject to petit mal attacks of nocturnal epilepsy for three or four years before his encephalitic condition. These attacks could be aborted by simple measures.
3. Early in his acute illness he appeared to be in a dream state or delirium. He kept his eyes closed and grunted like an animal. At that time his temperature was normal, and this without the cerebrospinal fluid findings, at first suggested an hysterical state. He later became distinctly unclear and disoriented. All these irritative symptoms preceded the lethargic state.
4. The substupor or lethargy did not appear until the neurologic signs became marked, more particularly eye signs, diminished knee reflexes and bladder symptoms. From this low grade stupor he could be aroused up to the last day or two, when the terminal coma set in.

CASE 4.—*History*.—H. H., a white man, a Russian Jew, aged 34, married, a sign painter, whose history was unknown to the wife, was negative according to the patient. He was born and educated in Russia. He had been in this country ten years, and spoke English unusually well. His best wage was forty dollars a month. His previous medical history was unimportant except for appendectomy about June, 1918, and influenza in the winter of 1918-1919.

Make-Up of the Patient.—He was married in August, 1919. He had neurotic tendencies before and after marriage. He was known to his wife only three months before marriage. Then and shortly after he expressed ideas of impotency; he was frequently unable to accomplish orgasm. Several months before his present illness he saw several physicians in regard to this condition.

Present Illness.—The patient was first seen on Feb. 10, 1920. For a few weeks before he had complained of so-called "neuritis," with pains at the back of the neck, and at the outer aspect of the right arm. Repeated examinations revealed nothing important, except an enlarged turbinate; ethmoidectomy was performed and later the sphenopalatine ganglion was injected with alcohol; following this the patient seemed to improve for a short time, but his former symptoms returned, and when first seen he complained of various pains localized in the right shoulder, the right mastoid region, at the vertex of the cranium and in other places. He described the pain as being sharp and like that of a hammer beating him every few minutes.

Examination.—Feb. 10, 1920: A neurologic examination from an organic point of view appeared to be entirely negative, except for an exaggeration of all the deep tendon reflexes. There was a marked glove and upper arm analgesia and anesthesia. This was less marked in the lower extremities. Hemihyperalgesia of the right face, arm and trunk appeared at a later interval. The cranial nerves were normal. Vibratory and muscle tendon sense and electrical reactions were normal.

Mental Picture.—The patient was groaning and talking in a whispered voice. On being questioned, he said he did not realize that he was whispering. When his attention was distracted all evidence of pain seemed to disappear. On compression of the eyeballs, the patient said he saw various colors, flowers, trees, "now a castle, now a lighthouse with the sun as though it were going down."

On February 14 and 15 his condition was unchanged. He was put under hypnosis. During the second hypnotic state he described a journey through Arabia. He stoutly denied all memory of falling to sleep. During the waking state he talked spontaneously about his sexual impotence, and showed lack of insight when he could not be convinced that he could have a child (his wife was then pregnant) and still be impotent. From February 10 to 15 his temperature was usually normal, occasionally being slightly subnormal, and on one occasion it was 99 F.

Apomorphin had only a slight effect in doses of $\frac{1}{10}$ grain three times daily. This was directed to jerking movements of the abdomen which seemed to be within the patient's control on and after February 13. On the 16th his temperature began to rise gradually. A spinal puncture was performed with the following results: cell count, 20; colloidal gold, curve flat; globulin, negative, and Wassermann reaction negative. On the same day he began to develop visual and kinesthetic hallucinations. He thought his bed was wired with electricity and imagined that chickens were being hatched under his arm pits

and under his pillow. He also developed suspicious tendencies against a brother who was summoned from New York.

On February 20, Dr. Albert M. Barrett and Dr. Carl D. Camp were called in consultation from Ann Arbor. At that time both knee reflexes were doubtfully obtained, there was a noticeable horizontal nystagmus, a slight ptosis of both lids and a distinct bilateral weakness of the sixth nerve. The nurse reported that he was constantly masturbating or manipulating the genitalia. The nurse also reported that the patient rehearsed in detail to his brother all that was said at his bedside by the physicians.

His case was looked on as one of so-called lethargic encephalitis.

Later Clinical Course.—Feb. 20, 1920, nasal examination was practically negative, except that there was a slight erosion of the anterior portion of the septum with dried bloody secretion. There was a slight deflection of the septum; the left nasal cavity was not obstructed. The blood count revealed 13,600 white cells. The temperature remained constantly elevated, ranging from 99 to 102 F., but averaging about 100 until April 20, when it became slightly subnormal and remained so for a week. It then returned to normal and remained so until the time of his discharge, May 23, 1920.

The jerking movements of the abdominal muscles persisted until about March 15, when they gradually began to lessen in intensity though they never disappeared entirely. At no time were there any bladder symptoms.

Mental Picture.—For a short time after admission to Harper Hospital, Feb. 16, 1920, the condition was much the same as that described above: He was restless, picked at the bed clothes, had hallucinations, was disoriented and at times incoherent. This condition gradually disappeared until about March 1, when a substuporous or so-called lethargic state appeared and continued for about a week. He could, however, at all times be aroused sufficiently to answer questions.

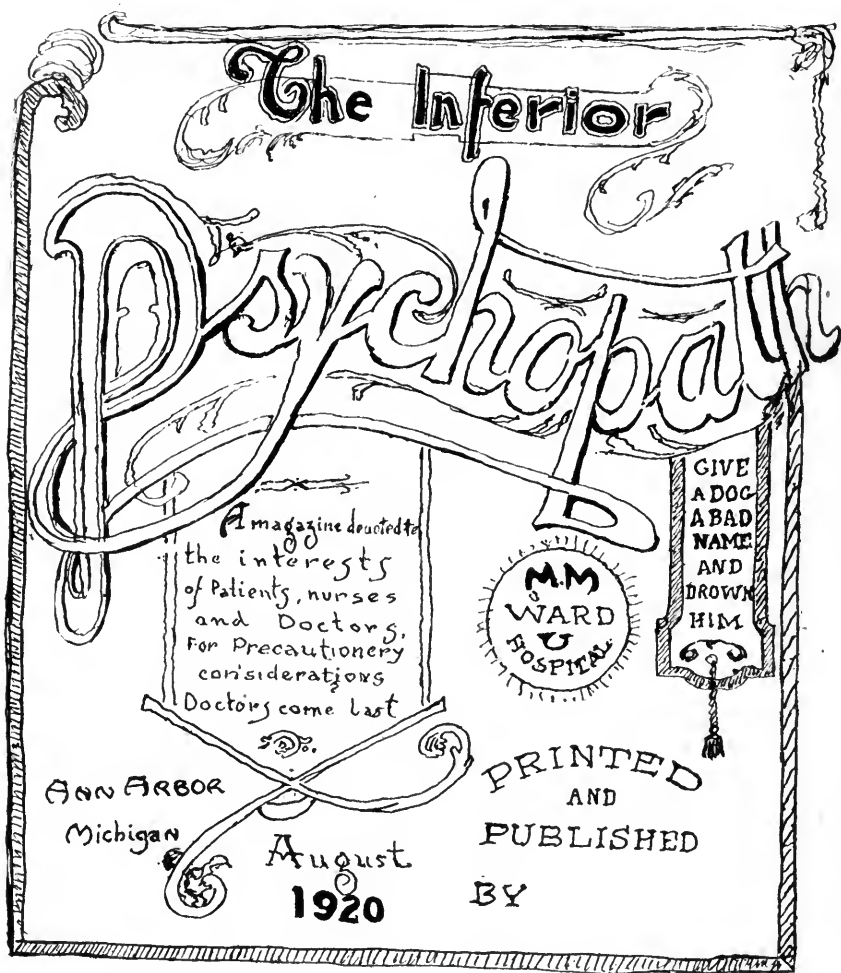
On or about the middle of April, two months after the acute onset, the patient began to have insight into his condition, but there was a distinct amnesia for the period of irritation or delirium and for the period of substupor. On April 18, he wrote to Dr. Mc.: "Having emerged from the world of shadows—a world all of its own, with its own laws, where the glimpses of reality that penetrate the mind serve only as material to draw the most weird pictures, and being now on the road to recovery, I take this opportunity to thank you . . . for calling on me at the hospital . . . although I don't remember it . . . people tell me about it . . . and last, but not least, I thank you for giving me Dr. J.—to whom I have so much to be thankful for."

The period of amnesia dated back to the time of the first lumbar puncture before his admission to Harper Hospital.

Though insight had returned, and the patient's mind was clear, he continued to show marked psychoneurotic tendencies. He seemed to delight in localizing the areas in which he stated he still had pain. He would point to one knee and say "I have pain here; but that is not all. My pain must be symmetrical, and so I have pain here," pointing to the same spot on the opposite knee. He would do this for almost all portions of the body. He would frequently speculate and dilate on his experiences and symptoms during his illness, making it appear at times that his amnesia was not complete. Whenever any mention was made about its being almost time for his discharge from the hospital, he would become querulant and complaining. He was seen once or twice in the outpatient department after his discharge, and on each occasion showed a lack of initiative, dilated on his various bodily weaknesses and

begged to be readmitted. He returned to work but only for a few days. He seemed helpless at home and continued to complain until he was finally admitted to the University Hospital at Ann Arbor on an order of the court. He is still a patient at that hospital.

That the psychopathic tendencies exhibited before his illness have not disappeared with convalescence is evident from the following interesting katamnesis. While awaiting the order from the court for his admission as a public



Cover page of "magazine" written by the patient in Case 4. This cover was designed and drawn by the patient after recovery.

patient to the University Hospital, it was necessary to detain him in the Psychopathic Ward at the Detroit City Receiving Hospital. By some chance he saw his chart or heard notes read from it and caught up the words "psychopathic inferiority" relative to his own case. Under date of Sept. 6, 1920, he wrote to Dr. Mc. a detailed letter, in the form of "A Magazine Devoted to the Interests of Patients, Nurses and Doctors. For Precautionary Consid-

erations Doctors Come Last." It was painstakingly printed by hand in pen and ink script with an illustrated cover and three pages of text. An exact reproduction of the cover is shown on page 158. A few excerpts from the "magazine" may be of interest. Evidently he no longer thanks Dr. Mc. for giving him Dr. J.

"Dear Dr. Mc.:

"Now, that I have several painless hours every day, and can sit at the writing table like a man, I wish to let you know that my wife gave birth to a baby girl.

"She is now nine weeks old . . . very beautiful and is an exact copy of me (there is no discrepancy in it, for I was nice looking when I was a baby) yours sincerely, H— H—, the individually psychopathically inferior following encephalitis lethargica.

"No, Doctor, I did not give myself deliberately such a name, it is a bull conceived in an unfortunate hour by Dr. J—. With this name, he sent me for a few days to the Receiving Hospital when they locked me up with insane criminals. When I asked the nurse . . . for a pillow . . . she told me to cut it out, and pointing to the history of my case, . . . she said that I am insane . . . and simulating pain because I want everybody to serve me . . . although she did not say it, but what she meant was probably that I am an egomaniac or afflicted with mania grandiosa. . . . Since I awoke from that long sleep, I have not spoken one word which would justify such a diagnosis. I cannot have anything against the deductions of the science of psychiatry, and I would not worry so much and it would not be so painful if some one else did it, but Dr. J— who has done for me so much, who has literally wrested me from the jaws of death, of him I did not expect this. And then I don't understand what it means, 'Inferior psychopath;' this implies the existence of 'superior psychopaths.' What do they look like?

"I know I was irrational during the first period of my illness; I don't know what the future holds for me; but at present am Sane. Or is it the fact that the cause of the pain is in the brain that made him call me that name? then why inferior? and would not 'Pathologic brain' or some other term do?"

This letter apparently indicates that the patient's condition is approximately what it was before his illness: he is an odd type with neurotic traits thrown into stronger relief by the physical after-effects of his earlier toxic state.

Summary of the Mental Picture of Case 4:

1. Russian Jew, fairly well educated, of distinct neurotic type.
2. Fears of sexual impotency before and after marriage.
3. Toxic character of illness masked in its early stages by what appeared to be purely hysterical symptoms, including anesthetics, analgesias, easy suggestibility with successful hypnosis and other symptoms, as well as aphonia.
4. After the toxic character of the illness was manifest there appeared symptoms of irritation, mental uncleanness and hallucinations; ideas of influence, delusions, disorientation, masturbatory tendencies, picking at the bed clothes and other symptoms.
5. Still later appeared the so-called lethargic, or substuporous stage from which the patient could be aroused sufficiently to answer questions.
6. Finally the amnesia, partially questionable, and the tendency to defense mechanisms seen in his letters. The so-called "magazine" illustrates this well, and even suggests hypomanic and dementia praecox features.

In reviewing the four cases just reported there seem to be two outstanding features of the disease, one not limited to the disease entity before us, and the other possibly generally characteristic of the

disorder, although not strictly limited to it. Without attempting to relate these features to the pathology of encephalitis, and looking at the subject from a clinical point of view, these two features seem to be: one, the symptoms of the irritative stage, and the other the symptoms of the so-called lethargic, substuporous or low grade stupor stage. That of the irritative stage is less characteristic and is a feature so frequently found in what is popularly styled "nervousness," "maniacal states," or "delirium" (dependent on the degree of irritation) of the various toxic psychoses. The most important irritative symptoms in the four cases just reported may be recapitulated in the order in which they present themselves:

Case 1: During illness there were marked emotional instability, unclearness or mental confusion, temporary loss of speech and amnesia for the delirium. After recovery emotional instability without deterioration appeared.

Cases 2: Terrifying dreams, insomnia, distractibility, mild kinesthetic hallucinations, failing powers of concentration, a tendency for odd numbers and letters to fix themselves in his consciousness, and a mild degree of euphoria were present. He manipulated the genitals (this may be regarded as purely a physical, rather than a psychic irritation). After recovery there were no irritative symptoms.

Case 3: The patient was in a dream-state or delirium and made grunting noises like those of an animal. His eyes were closed (possibly photo sensitivity) and he gave evidence of unclearness and disorientation, an incoherent stream of thought, and delusions of a mild persecutory character.

As for the substuporous stage, all of the patients except the patient in Case 1 showed some degree of lethargy or "emotional paralysis."^{1a} All three patients could be aroused from this condition, with the exception of the patient in Case 4 during his terminal coma; all three showed some degree of so-called parkinsonian or ironed-out facies, and all had an amnesia for both the irritative and lethargic periods, less marked for the lethargic period as this projected itself in two of the cases, in milder form in the period of convalescence or recovery.

The patient in Case 2 four months after convalescence temporarily dozed off into a brief drowsiness during his work.

Two other interesting psychiatric elements present themselves in all four cases. 1. The patient either had a tainted heredity or had shown marked psychoneurotic tendencies before his illness. In Case 1 there was insanity in the ancestry and the patient was being reared in the atmosphere of divorce and other family difficulties. The patient in Case 2 was of a high-strung disposition and was temperamental.

^{1a}. Wechsler, S.: The Symptoms of Epidemic Encephalitis Structurally and Functionally Considered, New York M. J. **112**:175 (Aug. 7) 1920.

In Case 3 there was a history of petit mal epilepsy, with hysterical symptoms, and the patient in Case 4 was a neurotic of the nth degree with sexual obsessions, suggestible to the point of hypnotism and a more or less helpless type of individual.

2. The three patients who recovered showed some residuals of the acute illness long after convalescence set in. One was emotionally unstable, another had brief periods of drowsiness and the previous tendencies of the fourth were highly accentuated. He imagined that he was the victim of ill-directed medical classification, and spent his time in writing letters not unlike those of a hypomaniac. He is rapidly becoming institutionalized and it is questionable whether he will ever again properly maintain himself and his family.

This last point, as far as the variable symptoms of the disease are concerned, is probably the most important. In short, the mental picture of the irritative stage as well as that of the post-convalescent stage, with residuals, is determined by the individuality of each patient. Consequently, no two cases are alike from the psychiatric point of view, particularly when analyzed relative to the irritative symptoms. The situation here is much the same as it is in general paralysis, dementia praecox and the other psychoses. Each patient has his own background on which the changing features of his acute illness are thrown and thus make the psychic element unique.

REVIEW OF LITERATURE

Although epidemic encephalitis, as such, has been accorded much attention in the literature, particularly during the past year, yet little, beyond casual mention in the better reviews and the occasional report of isolated cases, has been contributed on the more essentially psychiatric phase of this disorder, notwithstanding the undoubted importance of such an analysis.

On the peculiar lethargy or apathy so characteristic of the disease and responsible for its original designation, lethargic encephalitis, by Economo,² in 1917, much comment has been made. This state has been variously characterized by different observers as cerebral torpor (Cruchet³), or somnolence (Netter and Sainton⁴), or stupor with normal rationality (Tilney and Howe⁵), and seems to have varied, in degree, from mere listlessness or mental inertia, to frank coma,

2. Economo: Verein f. Psychiat. u. Neurol. in Wien., April 17, 1917.

3. Cruchet, R.: Bordelaise Conception of Encephalitis Lethargica, New York M. J. **112**:173 (Aug. 7) 1920.

4. Netter, A., and Sainton: L'encephalite lethargique. Presse méd. **26**:487 (Sept. 23) 1918.

5. Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, Paul B. Hoeber, 1920, pp. 85, 20, 60, 50, 96.

with catatonic features. The condition, on critical analysis, seems not to have been coma or even true stupor as it appears to have been generally possible, on proper stimulation, to recall the patient to objective consciousness for brief periods, during which, beyond in some cases a certain retardation or lengthened reaction time, there was but little evidence of mental disturbance or impairment; although, as suggested by Burr,⁶ it is questionable whether in the absence of external stimuli there was actually any mediation of mental processes or any emotional tone. Wechsler^{1a} suggests resemblance to an emotional paralysis, and Alexander and Allen⁷ include reference to it as an emotional stupor as does also Kennedy,⁸ who has characterized such cases as intellectually keen but emotionally stupid. The term pseudo-stupor, as suggested by Burr,⁶ seems well in point although it is questionable whether some such designation as substupor might not be more suitable.

Wechsler^{1a} suggests that conceivably this state may have a thalamic basis and mentions the rather doubtful possibility of basic pituitary or pineal influence. On the whole, however, as noted by Burr⁶ and Tilney and Howe,⁵ the condition seems more probably but a manifestation of the generalizing nature of the condition, as is borne out also on consideration of its basic pathology (Hammes and McKinley⁹). Russell¹⁰ thinks that the somnolence depends entirely on a lesion in the posterior part of the pons near the aqueduct of Sylvius, thus interfering with the flow of the cerebral fluid in the cerebral hemisphere to the spinal canal and resulting in an acute hydrocephalus.

The so-called catatonic features—catalepsy, flexibilitas cerea, parkinsonian facies and the other features—have been reported by most observers, notably by Bond,¹¹ Hammes and McKinley,⁹ Wilson,¹² Hall,¹³ Kennedy,⁸ Batten and Still,¹⁴ Tilney and Riley,¹⁵ Tilney and Howe,⁵

6. Burr, C. W.: Case Report, Trans. Philadelphia Neurol. Soc., Feb. 27, 1920; Arch. Neurol. Psychiat. **4**:105 (July) 1920.

7. Alexander, M. E., and Allen, H. E.: Lethargic Encephalitis, Arch. Neurol. & Psychiat. **3**:485 (May) 1920.

8. Kennedy, F.: Epidemic Encephalitis with Stupor, Med. Rec. **95**:631 (April 19) 1918.

9. Hammes, E. M., and McKinley, J. C.: Lethargic Encephalitis, Arch. Int. Med. **26**:60 (July) 1920.

10. Russell, C. K.: Study of Epidemic Encephalitis Based on Seventeen Cases with Two Necropsies, Canadian M. J. **10**:696 (Aug.) 1920.

11. Bond, E. D.: Epidemic Encephalitis and Katatonic Symptoms, Am. J. Insan. **76**:281 (Jan.) 1920.

12. Wilson, S. A. K.: Epidemic Encephalitis, Lancet **2**:7 (July 6) 1918.

13. Hall, A. J.: Epidemic Encephalitis, Brit. M. J. **2**:461 (Oct. 26) 1918.

14. Batten, F. E., and Still, G. F.: Epidemic Stupor in Children, Lancet **1**:636 (May 4) 1918.

15. Tilney, F., and Riley, H. A.: Epidemic Encephalitis, Neurol. Bull. **2**:106 (March) 1919.

and Bassoe.¹⁶ The exact mechanism concerned in the production of the muscular hypertonus constituting this feature has apparently not yet been definitely determined. Thus Tilney and Howe⁵ seem to feel that it is due essentially to extrapalliospinal involvement, while Wechsler^{1a} questions whether this sign can be explained wholly on the basis of extrapyramidal involvement and mentions the possibility of a true cerebral condition, an interference with cerebral functioning, with a resultant setting free of uninhibited unconscious activity, as found in dissociative epilepsy.

The possibility of the existence of a definite or characteristic psychosis is deprecated by Wechsler, who feels that findings suggestive of such a state are but manifestations of an underlying infection or toxic delirium syndrome. Tilney and Howe, in the inclusion of epileptomaniac and acute psychotic types in their clinical classification, seem to bear out this point of view, as do also observations by Wilson.¹² Cases studied by Hammes and McKinley⁹ showing delusions, hallucinations and temporary disorientation, and by Cruchet,³ showing disorientation and paraphasic features, may be construed on the same basis.

On the other hand, Climenko¹⁷ reports a case of definite Korsakow psychosis and Leiner,¹⁸ Hammes and McKinley, and Wilson remark the occurrence of Korsakow-like syndromes.

Leiner mentions two apparently frank cases of manic depressive psychosis, one of the cyclothymic type. Climenko also indicates this possibility, as an end point to the characteristic euphoria which he frequently found to precede the acute phase of the disease. This euphoria, he finds, shows a definite tendency to become a hypomania, as the disease progresses which, in turn, may become a truly manic condition. He further indicates the possibility of the development, in some subjects, of an excited-depression as opposed to a hypomania. Euphoria has been noted in addition, by Tilney and Howe,⁵ Schwartz,¹⁹ and House.²⁰

Wilson² reports a case in which the patient developed a typical witzelsucht.

16. Bassoe, P.: Epidemic Encephalitis (Nonna), J. A. M. A. **72**:871 (April 5) 1919.

17. Climenko, H.: Encephalitis Lethargica, New York M. J. **111**:531 (March 27) 1920.

18. Leiner, J. H.: Encephalitis Lethargica, New York M. J. **111**:179 (March 27) 1920.

19. Schwartz, S.: Encephalitis Lethargica, New York M. J. **112**:6 (Aug. 7) 1920.

20. House, W.: Epidemic (Lethargic) Encephalitis, J. A. M. A. **74**:372 (Feb. 7) 1920.

The available literature apparently indicates that psychiatrically considered the clinical picture in epidemic encephalitis is still somewhat indefinite. Nevertheless, there appears to have been definitely determined the characteristic initial pseudostupor or substupor (which may be preceded by a euphoric phase) distinguished by marked depression of the emotional field, often with catatonic features. In addition, as a function of the generalizing character of the infection and the reaction to it, there is evidence strongly suggestive of infection, toxic delirium or toxicorganic psychosis, which in some cases appears to simulate strikingly certain other common, noninfectious psychoses.

In the latter regard, although no specific mention has been made, there is no reason to suppose that a latent psychosis may not be precipitated as a result of the infection, as has been determined in the case of infectious processes, notably epidemic influenza.

CONCLUSIONS

1. The psychiatric phase of epidemic encephalitis has not received the same study and attention as has the neurologic aspect.

2. Looking at the disease from the psychiatric aspect, there seem to be two stages—the irritative stage in which the mental picture is not unlike that of other noninfectious psychoses, and the period of “low grade” stupor, lethargy or, better still, the substuporous period. The former may be marked by euphoria, hypomania, depression, hallucinations, paranoid stages, or even the Korsakoff syndrome, and frequently there are features that simulate those of severe hysterical excitement. The latter is rather an extreme apathy from which the patient may be roused; there is, as it were, a paralysis of the emotional field, frequently accompanied by masklike or parkinsonian facies. This apathy or stupor in some cases amounts almost to a catalepsy, but it should be remembered that it is possible, except in the terminal stage, to rouse the patient.

3. The keynote, if we may use this figure, is that the mental condition is variable, this condition being an infectious-toxic one, the whole dominated at some time by an apathy or substupor in which the personality is fairly well preserved but blurred by torpor.

4. The authors' four cases bring out these interesting features of the disorder:

- (a) The symptoms during the irritative stage reflect the patient's make-up previous to his infection.

- (b) There is an amnesia for the irritative period. In these two respects, the disease is characteristic of a toxic delirium.

- (c) Each patient experienced the substuporous stage except the first, a child of 9. This patient was not seen until some time after the onset.

(d) There are mental residuals in all patients that recovered, either similar to those noticed in the person before his illness, or else highly aggravated. These may gradually pass away, or become progressively more pronounced. The last result is true of Cases 1 and 4.

5. It may not be dogmatically maintained that the disease can be diagnosed from the psychiatric picture; the substuporous stage, however, is strongly suggestive of the disorder, and when found in conjunction with spasmodic twitchings, lost knee reflexes, bladder disturbances, cranial nerve palsies and other symptoms is almost if not truly diagnostic.²¹

21. Since the manuscript for this paper was submitted, two articles have appeared: one, in the November issue of the *ARCHIVES OF NEUROLOGY AND PSYCHIATRY*, by L. Archambault. He lays emphasis on the generalizing nature of the infection and speaks of one case in which there was a paresis-like picture. The other is by I. Abrahamson: "Mental Disturbances in Lethargic Encephalitis," *J. Nerv. & Ment. Dis.* 7:193 (Sept.) 1920. He also speaks of the generalizing nature of the intoxication and of the Korsakoff-like pictures in the disease.

A CASE OF ALTERNATING HEMIPLEGIA (MILLARD-GUBLER), WITH REMARKS ON THE PATHS OF THE PUPILLO-DILATOR AND VESTIBULO-OCULAR FIBERS IN THE BRAIN STEM

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Sharply defined anatomic lesions of the brain stem have recently acquired a new interest in so far as they are now able to furnish us exact information as to the course of those fibers which come from the upper part of the mesencephalon and extend to the centrum cilio-spinale. Irritation of these fibers produces psychic as well as reflex dilatation of the pupils. Information is also furnished regarding the path of those other fibers that transmit the irritation from the vestibular apparatus to the oculopontile nuclei. The following case is of interest in this connection.

REPORT OF A CASE

History.—Felipe de Ll., 31 years old, an engineer, complained Oct. 23, 1918, of reduction of visual acuity. He had always been healthy and strong. There were no family diseases. He denied syphilitic infection. When he was 11 years old, he fell from a height of 3 meters, on his cranium; as a consequence of this accident, there was a small nonadherent cicatrix of the skin in the right parietal region.

Since January, 1918, he had observed indications of metamorphopsia in both eyes, and reduction of his visual power. On June 21, 1918, he suddenly became ill, with symptoms of a left-sided hemiplegia, including the left side of his face, his arm, trunk and leg; at the beginning of this attack he was unconscious for several hours.

Examination.—Examination Oct. 31, 1918, revealed a pale, young, strong, well-nourished man, who complained of headache in the region of the right fissura centralis. The urine contained a trace of albumin (0.08 0/00), $\frac{1.200}{1.019}$ no sugar and no casts. The Wassermann reaction of the blood was negative. Percussion of the cranium did not produce pain. There was no Romberg sign or symptoms from the cerebellum. The tendon reflexes were normal on the right side, but seemed to be a little diminished on the left side. There were no indications of contracture and no Babinski reflex. The muscular force of the left arm and left leg were undoubtedly reduced, as well as the sensibility. The pupils were equal in both eyes and of medium width; they reacted well to light and accommodation; there were no anisocoria, diplopia, nystagmus, or reduction of the eye movements. Ophthalmoscopically the disks appeared blurred; they showed no prominence, and the entire fundus was covered with small white spots as in albuminuric neuroretinitis. The visual field showed a slight reduction of color sensation in the temporal half of the left eye, being otherwise normal.

Dec. 23, 1919: The ophthalmoscopic alterations had receded somewhat; otherwise the status was exactly the same as at the time of the previous examination. Examinations of the blood showed negative Wassermann reactions; and many examinations of the urine disclosed no evidence of nephritis. An examination of the cerebrospinal fluid showed a negative Wassermann reaction and a positive globulin reaction (Noguchi), with slight increase in quantity. The original diagnosis of neuroretinitis albuminuria could no longer be maintained. An examination of the vestibular apparatus by Dr. Fernandez Soto showed:

1. Slight reduction of the hearing of the right ear on account of cicatricial otitis (the lesion was caused by an insect in his youth).

2. Both acustici proved to be normal.

3. No spontaneous nystagmus, but spontaneous past pointing of the left arm toward the right. Examination on the turning chair revealed: in different investigations, slight reduction of the amplitude and duration (18 seconds) of the normal nystagmus from the horizontal canals, normal past pointing and vertigo. Ear stimulation with warm water, for the examination of the vertical canals, revealed a complete absence of nystagmus, past pointing, and vertigo; on changing the position of the head (horizontal canals) a reduced nystagmus, past pointing and vertigo appeared. There existed, therefore, a complete inexcitability of the vertical semicircular canals, and hypo-excitability of the horizontal canals. The patient showed no signs of labyrinthine disease (spontaneous noises, vertigo or deafness).

Jan. 21, 1920: On the entire left side of the patient's body (face, arm and leg) a well-marked thermoanesthesia appeared; a few days before he had an attack of dysarthria lasting for some minutes; there was no Babinski reflex.

March 8, 1920: While in the toilet, he suddenly lost consciousness and fell to the floor. He showed a flaccid paralysis of the left arm and left leg (but not of the face) and anesthesia of the entire left half of the body (including the face). The anesthesia could be observed especially well during sleep; one could prick him with a needle on the left side to the point of drawing blood and he showed no reflex defensive movement and did not waken; the reflex defensive movement was marked on the right side. Insensibility extended exactly to the middle of the nose and was most marked when the patient was awake, for temperature and pain and less marked for touch.

March 10, 1920: The patient complained of violent pains and pronounced hyperesthesia in the whole region of the right trigeminus.

March 18, 1920: The patient showed a total peripheral paralysis of the right facialis.

April 17, 1920: Ophthalmoscopically the patient showed in both eyes a well marked choked disk of 3 to 4 diopters. The visual field was normal. The trigeminal pains had disappeared. Vomiting, vertigo, cerebellar symptoms and anisocoria were not present. He had only paralysis of the right facialis and paresis and hypesthesia of the left arm and leg.

April 27, 1920: During the four days before death the patient again suffered pains in the right trigeminus, which increased to such violence that he attempted suicide. On April 27 he felt much better; he arose, and after taking a short walk, he fell, began to perspire copiously, and died within a few minutes with Cheyne-Stokes respiration. Necropsy examination was refused.

Summary.—A young, strong, nonsyphilitic man, in whom no signs of renal diseases could be traced with certainty, began to complain of metamorphopsia and loss of vision, and showed ophthalmoscopically the appearance of neuroretinitis albuminuria. A complete left-sided hemiplegia (face, arm, leg) receded, leaving only a marked anesthesia of the whole left side, more pronounced for temperature and pain. There were no signs of contracture, Babinski reflex, or exaggeration of the tendon reflexes. An examination of the ear failed to disclose disease but showed a complete lack of the function of the vertical semicircular canals and a slight reduction of the function of the horizontal semicircular canals. The patient did not have the signs of brain tumor. As time passed complete right-sided paralysis of the facialis and a marked hyperesthesia of the right trigeminus with spontaneous pains developed, added to the left-sided slight disturbance of motility and strong alteration of sensibility. The neuroretinitis albuminuria developed into a typical bilateral choked disk, and the patient died suddenly with symptoms of vagus death.

Necropsy having been refused, cause and localization of the anatomic lesion could not be ascertained with the desired exactness, but we may deduce the following:

The lack of casts in the urine, its normal specific weight and its normal quantity tend to show that a chronic renal malady did not underlie the disease. We have many observations in the earlier ophthalmologic literature showing that the most experienced ophthalmologists pronounced the diagnosis of "nephritis chronica" on account of the fundus picture, while the necropsy failed to discover any disorder of the kidneys but proved the presence of a brain tumor. The whole course of the disease, the developing of a typical choked disk, the positive globulin reaction in the cerebrospinal fluid, the development of a crossed hemiplegia and the sudden death under the symptoms of Cheyne-Stokes' respiration, indicate that we were dealing with a slowly advancing process in the brain stem, probably a tumor, or perhaps a tuberculum.

With greater exactness we can deduce the location of the lesion. The reduction of the sensibility in the whole left side of the body (face, arm and leg) shows an interruption of the lateral fillet between its crossing in the medulla oblongata and its termination in the thalamus opticus; it further shows that this lesion must be located on the right side of the brain stem and higher up than the entrance of the fibers of the trigeminus after their crossing, i. e., in the middle third of the pons. The right-sided peripheral paralysis of the facialis and the right-sided hyperesthesia of the trigeminus indicate, furthermore, a lesion on the right side of the middle part of the pons. The fact that the sensation of pain and temperature alone was completely abolished on the left side of the body and that superficial sensibility was only reduced, together with a peripheral palsy of the facialis without disturbance of the abducens; also the normal functioning of both acustici and the dissociated lesion of the vestibular fibers, and the flaccid character of the left-sided hemiplegia, show that there was a sharply defined anatomic lesion on the right side of the brain stem situated in the upper and external part, limited ventrally by the pontile nuclei, laterally by the vestibular nerve and mesially by the abducens and the mesial fillet; it did not ascend to the floor of the fourth ventricle, because the nucleus of the abducens and the fasciculus longitudinalis posterior were not altered in their function. The lesion included the descending root of the trigeminus (spontaneous pains on the right side

of the face) and destroyed the lateral fillet, the right pyramidal fibers dispersed between the pontile nuclei and the exit of the right facialis. On the right side the function of the facialis and trigeminus were abolished; on the left side, the greater part of the sensibility and the motility of the arm and leg were abolished. This syndrome has long been known as *hemiplegia alternans* (type of Millard-Gubler), and it would scarcely be worth while reporting it, were it not for three special points.

ABSENCE OF DEVELOPMENT OF CONTRACTURE

Notwithstanding that the patient showed twice, with an interval of about two years, a disturbance of the right pyramid, the paralysis always had a flaccid character and no signs of contracture developed. An interruption of the supranuclear motor path is followed as a rule by a spastic paralysis, and even if it completely recedes, an exaggeration of the corresponding tendon reflexes remains, and a positive Babinski reflex develops. The signs of contracture develop at the earliest after fourteen days, but as a rule from one to three months after the attack, and they fail to appear only in the rarest exceptions. Explanation of the developing of the cerebral contracture has been attempted, and careful investigations have shown the interruption of the pyramidal tract and the integrity of the posterior roots to be the essential conditions. Both conditions were present in my case, and nevertheless the reflexes of the left side were rather diminished. Of the different theories, that of van Gehuchten is best suited to the present case; it holds that the contracture is caused by the continuance of the functioning of those fibers which connect the pontile nuclei with the cerebellum (*fibrae cortico-ponto-cerebello-spinales*) through the middle cerebellar pedunculus.

LACK OF LESION OF THE PUPILLO-DILATING FIBERS

During the whole course of the disease I paid special attention to the observation of the pupillary reaction in order not to overlook the appearance of anisocoria. We know from many observations that a lesion of the brain stem is sometimes followed by a homolateral miosis, and therefore lesions of the brain stem are apt to illustrate to us the exact topographical course of the pupillary fibers. If we wish to determine their situation in the brain stem, we must look for the following points: (a) height and latitude of the lesions, and (b) crossing of the fibers.

(a) During the entire course of the disease both of the patient's pupils were equal and of medium size. This agrees with other symptoms that indicate a lesion in the tegmentum of the pons not having reached the floor of the fourth ventricle. Déjerine became convinced, through his anatomic investigations, that the sympathetic fibers run through

the upper layers of the substantia reticularis, medially of the root of the trigeminus and laterally of the middle of the brain stem. In a previous article I expressed the opinion¹ that the sympathetic fibers are responsible for the pupillo-dilatation and that they run down from the mesencephalon to the centrum cilio-spinale, together with the fasciculus longitudinalis posterior which is situated just on top of the substantia reticularis. Spiller² gave some further instances showing that the lesion of the brain stem can be followed by a homolateral miosis, and in his cases the sympathetic fibers have been interrupted by foci of destructive nature. In consideration of this point, tumors like gliomas which entangle the fibers are less apt to give information than tuberculous foci that destroy them. As we have not yet many observations of lesions of the brain stem which especially consider the localization of the pupillo-dilating fibers, it may be well to cite here, two further examples illustrative of the subject. F. Raymond³ gives an exact description of a case closely resembling my own: Paralysis of the facialis and of the abducens on the left side, and spastic paresis of the right arm, trunk and leg, together with cessation of all sensibility on the right side of the body (face, arms, trunk and leg) occurred; the patient showed no anisocoria. Another patient⁴ who had, on the right side, hemiparesis and hypesthesia of the arm, trunk and leg, and on the left side a paralysis of the facialis, abducens and masseter, together with anesthesia of the left side of the face, had a miosis without alteration of the pupillary reaction on the left side. The fact that the masseter was paralyzed shows that the lesion extended higher up in the level and extended more medially than the previously reported case and my own.

We may therefore conclude that the lesion of the brain stem will be accompanied by a homolateral miosis only when it reaches the highest and medial part of the substantia reticularis.

(b) Most authors believe, with me, that in man the sympathetic fibers do not undergo a crossing between the mesencephalon and the centrum cilio-spinale; some question that they undergo any crossing at all.

In favor of the first opinion that the pupillo-dilation fibers running down from the mesencephalon to the centrum cilio-spinale (which is

1. Lutz, A.: The Light Pupillary Reflex, Its Path and Its Abolition Called Immobility of the Pupil to the Light Reflex, and Report of a Case of Unilateral Argyll Robertson Pupil in which Consensual Reaction Existed in Both Eyes, *Arch. Ophth.* **47**:266 and 370, 1918.

2. Spiller: The Oculo-Pupillary Fibers of the Sympathetic System, *Am. J. M. Sc.* **100**:325, 1920.

3. *Maladies du systeme nerveux*, 1900, p. 63.

4. *Maladies du systeme nerveux*, 1903, p. 436.

the homologue of the motor cells of the anterior horns for the pyramidal system) remain uncrossed on the brain stem, are the above named reports on anatomic lesions, as well as the experiments of Levinsohn.⁵ Levinsohn cut the brain stem and the medulla-spinalis cervicalis in rabbits on one side and observed that this lesion was followed by a homolateral miosis. This miosis was always homolateral and not pronounced, but unmistakable, when he made the cut through the medial parts of the brain stem above the trigeminal entrance. Miosis was stronger and mostly homolateral, but sometimes also in a minor degree heterolateral after he had made the cut below the entrance and more lateral (descending roots of the trigeminus) miosis was never accompanied by a reflex immobility of the pupil. This developed only when he made the cut at a height between anterior and posterior corpora-quadrigenina. Levinsohn explained his experimental miosis by way of irritation of the pupillo-constrictor fibers, instead of by way of paralysis of the pupillo-dilating fibers. Therefore Levinsohn found in rabbits, as did Karplus and Kreidl⁶ in cats, that each half of the cervical cord conducts impulses from the brain stem to both cervical sympathetic nerves. In man, however, all our experience tends to show that the miosis is homolateral with a lesion of the brain stem. This shows us that the important experiments of Karplus and Kreidl made on cats and apes with regard to the subthalamic dilation center and its connection cannot be applied unreservedly to human pathology.

The question of crossing is more difficult to decide in the parts of the pedunculus situated higher up because of the lesion occurring at the same time in the fibers of the oculomotorius responsible for pupillo-constriction. For the following reasons we may conclude that they do not undergo crossing below the anterior quadrigenina: Brissaud⁷ remarks that an ophthalmoplegia externa pura is as a rule accompanied by a miotic pupil without alterations of the reactions. Cases of unilateral Argyll Robertson pupil, such as are reported by Tanzi,⁸ Salomonsohn,⁹ and myself,¹⁰ show miosis. The results of Levinsohn's experiments with rabbits, and cases of hemoplegia reported by Spiller, and the case of unilateral thalamic lesion of Ingham, referred to by Spiller in the same article, apparently point to this conclusion.

5. Beiträge zur Physiologie des Pupillarreflexes, Von Gräefes Arch. f. Ophth. **59**:191, 1904.

6. Karplus and Kreidl: Arch. f. d. ges. Physiol. **129**:138, 1909; 1910, p. 401; 1912, p. 109.

7. Maladies du système nerveux, 1895.

8. Riv. di patol. nerv., 1899.

9. Von Gräefe's Arch. f. Ophth. **54**:211, 1902.

10. Arch. of Ophth. **47**:266, 1918.

If we deduce from the foregoing that the pupillo-dilation fibers do not cross, in man, between corpus quadrigeminum anterius and centrum cilio-spinale, we are forced to search for the crossing higher up. For it is a general anatomico-physiologic law that every efferent motor system undergoes a crossing in its supranuclear path. We must further remember that there exists a direct, as well as a consensual, pupillo-dilating reflex in man; this indicates that a partial decussation of the pupillo-dilation fibers must occur higher up, and it is quite possible that this happens in the dorsal tegmental decussation of Meynert. It appeared logical to look for the crossing of the sympathetic fibers in the commisura cerebri-posterior, which Ramon y Cajal considers an optic reflex path; but we know, through the experiment of Harris,¹¹ that its cutting is not followed by miosis, and we must therefore conclude that at least the majority of the pupillo-dilating fibers do not take this course.

The question of where the sympathetic fibers have their source is as yet unsolved. Histologic researches of Ramon y Cajal show that the fasciculus bigeminalis connects the lenticular region with the corpora quadrigemina anteriora. Ramsay Hunt¹² came to the conclusion that these fibers of the ansa peduncularis originate exclusively from the globus pallidus, i. e., from those parts of the nucleus lenticularis which, in the realm of motion, plays the same important rôle, through control and regulation of automatic and associated movements, as the thalamus opticus in the domain of sensation, and both of which lost phylogenetically their early importance with the development of the cortex cerebri. Testut tells us that part of the ansa peduncularis enters the fasciculus longitudinalis posterior, which has perhaps for the basal motor ganglions the same importance as the pyramis for the cortical motor region. These pallidal fibers govern the principal movements of Munk, as do the pyramidal fibers with Munk's separate movements. Ramsay Hunt concluded that the fibers originating in the globus pallidus have a purely motoric function: this means that those which run down in the fasciculus longitudinalis posterior dilate the pupil. Consequently, their interruption would be followed by miosis.

Karplus and Kreidl⁶ determined by experiments with cats and apes, a center for pupillo-dilation, situated in the hypothalamus, near the dorsomedial portion of the foot of the cerebral peduncle, in the frontal part of the corpus subthalamicum. They could further show that the

11. Harris: Binocular and Stereoscopic Vision in Man and Other Vertebrates with Its Relation to Decussation of the Optic Nerves, the Ocular Movements and the Pupil Light Reflex, *Brain* 27:107, 194.

12. Hunt, Ramsay: Progressive Atrophy of the Globus Pallidus, *Brain*, 40:58, 1917.

irritation of this center in apes had a stronger effect on the heterolateral sympathetic cord. Their center corresponds, probably, to the corpus of Luys, limited beneath by the pedunculi cerebri, medially by the substantia grisea of the third ventricle, above by the thalamus opticus and laterally by the capsula interna. Ramsay Hunt considers it a derivative of the globus pallidus. All the subthalamic centers undergo, after the histologic search of Ramon y Cajal, a partial crossing; the fibers of the corpus of Luys cross just in front of the red nucleus (commissura de Forel). Ramsay Hunt also came to the conclusion that a degeneration of all the fibers originating in the large cells of the globus pallidus is followed by a syndrome of paralysis agitans, i. e., rigidity, tremor and loss of the automatic function of the corresponding somatic muscles. Another peculiarity of the Argyll Robertson pupil is that it remains not only immobile to light, but also loses the constant and automatic small oscillating movements which the Germans call "Pupillenunruhe"; the amaurotic pupil is also immobile on light incidence but never loses its "Pupillenunruhe," nor becomes miotic. The fact that an Argyll Robertson pupil is not influenced by a retinal process, e. g., that it remains miotic, although the patient becomes blind later by optic atrophy, indicates clearly that the Argyll Robertson pupil is not only produced by interruption of the afferent optico-sensoric path, but by still other etiologic factors. Furthermore, the Argyll Robertson pupil not only grows stiff, but its dilatation becomes difficult by reason of atropin. One can therefore consider the possibility that the peculiar stiffness of the Argyll Robertson pupil is caused by an interruption of striospinal fibers traveling in the fasciculus bigeminalis and their continuance in the fasciculus tecto-spinalis. Against such acceptance, we must say that bulbar as well as spinal miosis are as little followed by rigidity of the pupil as the amaurotic light immobility.

Nevertheless, we must accept the fact that the peculiar stiffness of the Argyll Robertson pupil is produced by a lesion of the strio-thalamo spinal system, because rigidity plus paralysis are as typical for lesions of the globus pallidus as is spastic paralysis for lesions of the pyramid. Perhaps it is produced by interruption of those fibers which enter in connection with the deepest layers of the corpora quadrigemina-anteriora. We may also remember here that we not seldom find in old people miotic and rigid pupils without loss of light reflex; this is perhaps caused by senile vascular changes in the lenticular region, whereas a lesion in the dorsal tegmental decussation conduces to the Argyll Robertson pupil, namely: to loss of light influence through interruption of the tectobulbar fibers,¹ to miosis through interruption of the tectospinal fibers, to a rigidity through interruption of the

pallidal fibers. If these deductions are correct, we are forced to accept that the crossing of the latter two fibers takes place higher up in Meynert's crossing than the partial decussation of the first named fibers. Also, we will find the following homology between the paths for pupillo-constriction and pupillo-dilation:

PATH FOR PUPILLO-DILATION	PATH FOR PUPILLO-CONSTRICTION
AFFERENT	
Skin corpuscle Spinal ganglion Nuclei of Goll and Burdach Sensible crossing in medulla oblongata Cells of the globus pallidus and corpus de Luys	Cones and rods } Bipolar cells } Retina Ganglion cells } Chiasma Nervus opticus Corpora quadrigemina anteriora
EFFERENT	
Fasciculus bigeminalis plus fasciculus tecto-spinalis	Fasciculus tecto-bulbaris
CROSSING OF MEYNERT	
Centrum cilio-spinale Ganglion, superior cervical Dilator iridis	Nucleus oculo-motorius Ganglion ciliare Sphincter iridis

DISSOCIATION OF THE FUNCTION OF THE SEMICIRCULAR CANALS

The patient does not show any signs of disease of the *nervii acustici*, but there is doubtless a dissociation of the function of the semicircular canal. The examination after the method of Baranyi, with warm water, discloses that the vertical canals do not react, either with nystagmus, vertigo, past-pointing or nausea; whereas the horizontal canals react in every way, though in a lesser degree, as shown by the reduced amplitude and the reduced duration of the experimentally provoked nystagmus. Such a dissociation of the semicircular canal can be caused, according to experience, only by a lesion of the brain stem. We know, through the systematic investigation of Jones,¹³ Mills and Weisenburg, that the vertical and the horizontal semicircular canals send their fibers through different paths to the bulbar nuclei, to the cerebellum and to the cortex. The horizontal canal fibers divide before the magnocellular Deiter's nucleus, and send their cerebellar fibers, which correspond to the unconscious muscular fibers of the body, through the inferior cerebellar peduncle; the vertical canal fibers go higher up into the pons (perhaps to the triangular nucleus of Obersteiner) and send their cerebellar fibers through the middle cerebellar peduncle. Our patient lacks from the vertical semicircular canals, not only nystagmus caused by the fibers which go to the ocular nuclei, but also vertigo caused by the fibers which ascend together with the conscious muscular sense and superficial sensibility through the medial lemniscus to the cortex,

13. Jones: *Equilibrium and Vertigo*, Philadelphia, J. B. Lippincott Co., 1919.

and which produce the sensation of vertigo by the difference of the conscient muscular sensation and the unconscious cerebellar muscular-tonus. The patient shows further a lack of normal past-pointing produced by the fibers which go to the cerebellum and cause the unconscious muscular-tonus. It must, therefore, be accepted that the fibers of the vertical semicircular canals are blocked in their totality. It would be quite easy to explain this loss of function if it were found only on one side, because all the other symptoms indicate anatomic lesion in the middle story of the pons just in the neighborhood of the entrance of the nervus vestibularis. Such a unilateral loss has been clinically observed by Jones¹³ in a man who, according to the opinion of Weisenburg, suffered from a typical lesion of the middle cerebellar peduncle: the left vestibularis was completely normal; the right one showed only the loss of function of the vertical semicircular canal. The patient in my case showed the loss of function on both sides, and the above deduced anatomic lesion cannot explain directly the loss of the second vestibularis, as we must expect from the cerebellar fibers that they ascend through the homolateral middle cerebellar peduncle. The only manner in which we can explain such bilateral loss is the following: The vertical fibers ascend higher up in the pons and run, therefore, a longer way beneath the floor of the fourth ventricle than the more numerous horizontal fibers; by their longer course they are more exposed to a blocking through an increase of the intraventricular tension. Already Wells P. Eagleton¹⁴ has pointed out that a reduced excitability of the vestibular apparatus of the apparently healthy side is produced by an increase of intracranial pressure. Jones came to the same conclusion as a consequence of numerous careful investigations. Both could observe that a decompressive operation reestablished the function of the vertical semicircular canals. An excellent illustration is one case observed by Jones:¹³ An apparently healthy man complained of headache; an examination of the vestibular apparatus revealed loss of the function of the vertical semicircular canals on both sides; irritation of the horizontal canal produced nystagmus but no vertigo; both acustici were normal and repeated neurologic examinations were negative. Some days later the patient died and necropsy disclosed abscess of the fourth ventricle. The exact investigation of the vestibular apparatus dates back only several years, since its introduction by Baranyi; we have, therefore, only a few observations on

13. Jones: *Equilibrium and Vertigo*, p. 124.

14. Eagleton, Wells P.: *Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin*, *Laryngoscope* **23**:592 (May) 1913.

13. Jones: *Equilibrium and Vertigo*, p. 58.

dissociation of the vestibular function. Jones, however, found thirty-eight among 700 cases on which he made clinical investigations, which perhaps indicates that in the near future numerous similar observations will be made. In all his cases the acustici were normal, the horizontal canals reacted, and the vertical canals showed a partial or complete unilateral or bilateral loss of function. We are not yet able to interpret exactly its bilateral loss. Eagleton, as well as Jones, could also have made the observation that tumors of the cerebellopontile angle block very early, through pressure on the brain stem, but only the function of the vertical semicircular canals of the opposite side is affected.

Therefore, the observation of the eyegrounds with a transition of neuroretinitis albuminuria in a typical choked disk, as well as the bilateral loss of function of the vertical semicircular canals, indicate that the patient in the present case suffered from an increase of intracranial pressure, which probably was produced by an intrapontile tumor causing a crossed hemiplegia and leaving intact the sympathetic pupillo-dilating fibers, because the lesion did not ascend high enough into the substantia reticularis.

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ENDOCRINE STIMULATION AS AFFECTING DREAM CONTENT

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REPORT OF A CASE

Following an attack of influenza in January, R. M., a single woman, 45 years old, with a history of previous good health, suffered from lassitude to an extreme degree. She was usually energetic and held a position that necessitated considerable mental and physical activity. Her history up to this time included the usual contagious diseases of childhood, pneumonia, bronchitis and influenza in the epidemic of 1918. The last attack of influenza had been a mild one, with only slight rise of temperature and moderate bronchial and gastro-intestinal symptoms. She remained away from work only two days, and then gradually resumed her usual routine though hampered by great fatigability. About two weeks after this partial recovery, the lassitude still persisting, she applied for treatment.

Miss M. was a stout, well-nourished woman with excellent color and a general appearance of good health. There was no anemia, no sinus involvement or other sequelae to her influenza. She ate and slept well, the cough had subsided, and the gastro-intestinal system was functioning normally. The lassitude was greatest in the morning when she found it extremely difficult to force herself to get up. After she had started to work she felt increasingly better until 4 or 5 o'clock when fatigue began, and she went to bed exhausted about 8. The blood pressure was taken in the early afternoon when the patient was at her best, and was found to be 90, systolic. It was probably much lower at times.

As Miss M.'s position required long hours of steady application it was necessary to restore her strength quickly, and a course of endocrine therapy was decided on as the best and shortest means of raising the blood pressure and increasing capacity for work. She was given one grain of the extract of whole pituitary gland each morning, beginning the latter part of January. For several days there was no change in her condition but by the end of a week or ten days the blood pressure had risen to 110, systolic, she felt better, began to have a return of initiative and was able to work her usual number of hours.

Dreams.—About this time Miss M., who had always slept soundly, began to have vivid dreams every night. Previously she had not been conscious of dreaming a dozen times a year, and then only after eating heartily just before retiring. She had always fallen asleep promptly, but after ten days of administration of pituitary extract, as soon as her head touched the pillow she began a series of delightful dream experiences. She began to anticipate going to sleep. The dream adventures were actually commonplace as she remembered them in the morning, and she was at a loss to account for the extreme pleasure they gave her and that she always awoke happy. Miss M. is of an active, cheerful disposition, and the few dreams she had had previous to this experience had usually been pleasant though characterless and hard to recall. During the pituitary period dreams followed each other in rapid succession

all night; she felt herself tumbling from one experience to another; and often they were so vivid as to awaken her—always with a pleasurable sensation.

The patient was fond of traveling and frequently spent her vacations this way. The dreams often took the form of a journey. One many times repeated, but always with varied surroundings and coming as a new experience, was that she was starting on her vacation—at the railroad station—sometimes alone, sometimes accompanied by a sister who has often taken trips with her, occasionally by a party of friends to see her off. But in all these dreams the sun was shining, the friends were laughing and joking; she was happy in anticipation of a pleasant journey. In several dreams the patient noticed that the usually dingy station was clean and bright, that the cars were new and freshly painted, the trainmen wore new uniforms and were smiling. Often it was not a vacation but starting to spend Sunday with her mother in the country, the dreamer intensely happy to be able to go. Less often she seemed to have reached her destination and to be wandering among new scenes and people. The latter dreams were much less distinct and the details not so well remembered.

Miss M. could not recollect having ever dreamed of colors until after she had taken pituitary extract, but in this whole series of dreams colors were bright and distinct; no one predominated over the others. One dream at this time was so vivid that it still seems like an experience of waking life. Miss M. was in the dining car of a railroad train crossing the plains between Chicago and the Rocky Mountains. She was certain of the locality, which she had visited several times. She was looking out of the car window at the flat lands enjoying in anticipation the mountain scenery she expected to reach next day. As in all of these dreams, the colors left the most distinct impression—the bright sunshine, the yellow fields of grain, the deep blue of the sky, the white clouds, the green grass. In the car the silver on the table glittered, the cloth was very white, the mahogany woodwork was almost cherry red. Her companion at the table was of so little account that in the morning she could not remember whether it was a relative or chance acquaintance of the journey. Only one dream in this series was of a different character. In this she was with her married sister, who has children, and her mother. Miss M. dreamt that she was sitting with her mother and sister and that she was expecting the birth of a child. She suffered no pain and the dream was principally occupied by a discussion with the married sister as to whether it were possible to have a baby so easily. The patient has seen women in labor and realized in her dream the unusualness of her experience. There were no details of the birth, but she woke happy and triumphant because she would have the baby with so little trouble. In this dream there was no thought of a father for the child, the patient and her mother and sister taking her condition as a matter of course. Miss M. is very fond of children and has often talked of adopting one.

The principal characteristics of this series of dreams were sensations of light and color—of action, going somewhere—and of pleasure in anticipating happiness to come. They were recalled by the patient as a succession of vivid pictures. Her memory of actual events was always in picture form to a much greater degree than the average. The predominant emotion of the dreams—hope—is characteristic of pituitary activity. The unreasoning depression of hypopituitary patients and their ready response to glandular therapy, their rapid return to a cheerful outlook on life, is in exact accord with the experience of Miss M.

Certain physical symptoms now supervened. By the time she had taken pituitary extract three weeks the patient developed a rather coarse intention tremor of the hands and her thumbs were occasionally sharply adducted. The latter symptom began a few days before the tremor, in the right thumb, later attacking the left one, and was very annoying, making it especially hard to hold a cup without overturning it. She was feeling so much better, however, that she did not report to her physician but continued taking the pituitary gland for a week or two longer. When she did present herself both symptoms were pronounced. The pituitary extract was discontinued, but as the blood pressure was still 110 and as the patient was easily fatigued, one grain of extract of suprarenal (the whole gland) was given each morning and a half grain at noon.

A week after the administration of pituitary extract was discontinued the tremor and adduction of the thumbs became less marked though the former recurred from time to time for several weeks. At about this time the patient suddenly felt well. She went to bed very tired as usual, but awoke in the morning able to get up immediately and begin the day. Until then she had found it impossible even to dress until at least an hour after her breakfast, which at this time included strong coffee. Her blood pressure was taken and found to be systolic, 120; diastolic, 75.

The dreams continued after the administration of pituitary extract was stopped; but a few days after beginning the administration of suprarenal gland they changed their character. They were less vivid and not so easily remembered, there were no colors, and the dreams were without exception unpleasant. Toward the end of the course of suprarenal extract the patient would wake two or three times during the night quite rigid, with a sensation of horror, her muscles so tense that they would ache for some time after waking. Usually the dreams were vague and only the emotions they called forth, fear or anger, would be clearly remembered. Occasionally she dreamt that she was awaiting news that she knew would be terrible. Twice she dreamt that she had violent quarrels. One dream of this character was so real that she awoke shaking with anger against a fellow-worker and could not sleep again for hours. In this dream the whole course of the quarrel, which was a verbal one, was vividly remembered though its cause was forgotten. Once she dreamed that she had committed some crime and was hiding from the police who finally captured her. In this dream she was not in her own identity, though who she was she could not recall, except that from the surroundings and events of the capture she thinks she must have been a man. The crime for which she was apprehended was likewise vague, something to do with a bank.

One of the most distinct dreams of this series occurred after attending a circus and seeing dwarfs. She thought that a horribly misshapen child was clinging round her neck, and that she was unable to loosen its arms. In this dream there was no sense of being strangled. She realized, while struggling fruitlessly to release herself, that it was only the mental horror of having so hideous a creature near her that caused her fear.

After taking the suprarenal gland extract for ten days the patient, who had always menstruated regularly, had a slight flow halfway between her periods lasting two or three days. As by this time she was feeling entirely well and was evidently profoundly stimulated by the endocrines, all medication was stopped. In about a week the dreams ceased altogether, becoming less frightful within two or three days after the suprarenal extract was discontinued. The blood pressure dropped to systolic, 110; diastolic, 70, and

the patient felt a slight return of her old lassitude. The only other symptoms at this time was the appearance of two or three small moles that persisted several months but had atrophied by the time this account was written.

Two weeks after the abnormal menstrual discharge and about a week after the dreams had ceased, the patient had a normal menstruation lasting six days as usual. The dreams recurred and continued through the entire period. They were all pleasant dreams, resembling those that occurred while she was taking pituitary extract. In the one most distinctly remembered she was in her bedroom dressing for some evening entertainment. As usual in these dreams she was very happy and decided to dress her hair elaborately to express this feeling. She arranged it with red and yellow ornaments, bright and glowing, standing out in a sort of halo round the face. She was pleased to see that her hair looked better than ever before. The rest of the family, when she left her room ready to start, exclaimed at the daring headdress and tried to persuade her to change it; but knowing it was becoming she refused, and the dream ended with her starting for her evening's pleasure triumphant in the consciousness that she looked her best.

During the next three months the abnormal menstrual discharge occurred with exact regularity halfway between the normal periods, growing less each month until it was only a stain. It was never accompanied by dreams. Each normal menstruation, however, was marked by a recurrence of agreeable dreams which grew less vivid and fewer each month until they ceased altogether. After the slight regression following the discontinuance of the endocrine administration the general health of the patient steadily improved and for the past two months she has felt perfectly well, with a blood pressure of systolic, 120; diastolic, 80; to systolic, 126; diastolic, 80. Her old capacity for work has returned.

SUMMARY AND COMMENT

In this case we have a woman who has seldom been ill enough to require treatment, aside from the acute infections; she once worked eleven years without losing a day through illness. She is overweight but otherwise in good condition; she has never suffered from any nervous disease. Interest in the case lies in the effect of stimulation of the endocrine glands on the dream content. Aside from the slight depression and apprehension caused by lack of strength in one who must work to live, the patient's mental and emotional condition was as usual. Yet after a purely physical disturbance with overstimulation of the sexual system, as the interference with the menstrual cycle showed, there appeared a series of dreams which, if their cause were unknown, would seem to point to a profound emotional disturbance.

The hypophysis, perhaps our chief stimulant to action through desire, was in this case augmented artificially. During the day there was little appreciable increase in its functions. The patient was overtaxing her strength at the time and noticed only a slight increase in energy while taking it, but at night—when there were no calls on the body beyond its diminished powers—the augmented pituitary gland gave symptoms of overactivity of its normal functions, which included ovarian stimulation. Thus the cause of the dreams was not psychic

trauma, but the result of too great pituitary activity which stimulated the ovaries and raised to the surface of dream consciousness some of the many unfulfilled desires present in us all.

The highly emotional, disagreeable dreams that followed those of frank desire on the administration of suprarenal substance, illustrate the action of this gland as the physical basis of fear and its kindred emotions, and these dreams are capable of the same explanation as the wish dreams. It is interesting to note that Miss M. was not emotionally affected by the endocrines during her waking hours and actually felt more cheerful while under the influence of suprarenal substance because, as her blood pressure increased, she was better able to work and ceased to worry about her health.

As living creatures increase in complexity and specialized organs appear the gonad system develops as the physiologic means through which the organism reacts to emotions. Pathologic overactivity of the various endocrine glands is capable of autonomously calling forth emotions without, or wholly disproportioned to, any foreign stimulus. In the case of Miss M., two of the glands controlling desire and fear were artificially augmented and the ensuing condition was the same as that produced when one or the other of these glands is over active per se. The picture is clearer than in most cases because the augmentation of one gland has not been caused, as it so often is, by the hypoactivity of another. This case illustrates through the dream the physiologic control over desire and fear, the two great factors in human conduct.¹ It is a good example of the purely physical origin of many dreams and the emotions behind them; an aspect of the subject that has been somewhat lost sight of in the fervor of Freudian interpretation.

104 East Fortieth Street.

1. Bailey: Desire and Fear as Factors in Human Conduct, President's Address, Transactions of the American Neurological Association, 1912.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The first annual meeting of the Association for Research in Nervous and Mental Diseases, held in New York City, Dec. 28 and 29, 1920, was very successful. The subject of lethargic encephalitis in its various aspects was discussed in about thirty papers. These articles, together with questions by members of the Commission on Scientific Work and others and the answers by the readers of the papers, will be edited and published by the commission.

The officers elected for the ensuing year are: president, Walter Timme, New York; vice president, E. W. Taylor, Boston; secretary-treasurer, Foster Kennedy, New York. Members of the Commission on Scientific Work, in addition to the officers, are: William G. Spiller, Philadelphia; Hugh T. Patrick, Chicago; Charles L. Dana, Bernard Sachs, Israel Strauss and J. Ramsay Hunt, New York.

The next meeting will be held in New York City during the Christmas holidays of 1921.

Abstracts from Current Literature

SOME MORPHOLOGIC DIFFERENCES IN SPIROCHAETA PALLIDA IN THE BRAINS OF GENERAL PARALYTICS, WITH ONE TABLE OF ILLUSTRATIONS. GEORGE SPRINGER, Arch. f. Psychiat. **61**:479, 1920.

The author summarizes the observations of earlier writers on the length and size of the spirochetes and on the length and thickness and mode of curving or bending of their spirals. He points out the necessity of more constant and methodic study of these conditions in order to establish some general laws in regard to the varieties and diversities of spirochetes found in the human brain in general paralysis of the insane.

Springer reports a case of typical paralysis which, however, from the early age of the patient at its onset might possibly fall into the class of juvenile paresis. In this case the spirochetes were present in great numbers, and the greater part were unusually long and slender, being from two to three times the length of spirochetes ordinarily seen in brains in general paralysis. The spirals, which generally show from 8 to 13 turns and occasionally as high as 20 to 24, in this case attained from 26 to 30 and even as high as 32 turns. The turn of the spiral was generally somewhat flattened not only in the middle of the organism, as commonly occurs, but throughout the whole length. While Noguchi gave 0.2 m. as the thickness of the thinner examples seen by him in contrast with his two other varieties at 0.25 and 0.3 m. in thickness, Springer's thin variety shows only 0.15 m. at the most. The spirochetes were so fine and so long that they gave the impression of a tangle of the finest of threads on a carpet. In addition to their extreme tenuity they underwent sharp bends and irregular windings. These appearances were not caused by shrinking of the tissues or other accidents of the tissues of the preparation since side by side with the sharply bent and irregular organisms others were seen of perfectly regular outline. Although flattened spirals were the rule in this case, small numbers of organisms were found scattered among those described which resembled the normal picture of the spirochete, with broad and thick turns of the spirals. But even in these forms the spirals were from 26 to 30 in number. They were found, as a rule, in thick groups or swarms of other spirochetes, and in the same localities short and thick forms were seen in small numbers and small round dark-staining bodies were also seen often attached to a spirochete. These small objects were from one to four times the diameter of a medium sized spirochete, and the author is certain they were not in any sense artefacts. In general, they were in contact with the shorter and thicker spirochetes and not with the long, thin variety. In thick clumps of spirochetes many were seen of normal length and size; in fact, the thicker the clump, the nearer normal in length, thickness and type of spirals were the spirochetes. The small balls may be permanent forms of the organism.

In some swarms of spirochetes large numbers were seen which looked extremely fragile, mostly fine pointed rod forms lying in a row; in their neighborhood were seen short, but still spiral forms which were made up of many little block fragments of varying size. Short spirochetes were also seen of normal form and medium size in thickness, but with very few turns of the spiral. The small fragments, however, would appear preeminently in such a

picture. In serial sections such a clump would show throughout a whole block, showing that they were by no means an accidental product. In such a swarm, many of the little balls spoken of were seen. Sometimes many individuals in such a clump did not stain normally, but were yellowish and brownish.

Rolled up forms were seen in moderate numbers and in general limited to the larger masses of spirochetes. They were rolled at one or at both ends; buds, ring and star forms were also seen. Whether there is any connection between the small round bodies and the buds, and whether the thick rings represent the union of two individuals which frequently has been seen in the form of a twisted loop is difficult to determine.

The medullary portions of the brain were free from the spirochetes. All layers of the cortex contained them, but the fine threadlike forms were most common in the more superficial layers. The greater number of swarms and clumps were found in the outer layers, but the fifth and sixth layers also showed a certain number of these conglomerations. Frequently considerable areas would show diffuse localization of the spirochetes only and frequently in small numbers. The parasites often lay in little coils of long threads, sometimes surrounding glia cells and more rarely, ganglion cells. With the larger masses, of which from one to two dozen would lie in a microscopic field which showed a practically diffuse localization, more rarely thick sheaths around the capillaries were seen, as described by Jahnel.

The author claims no priority for the foregoing descriptions, but considers that every well studied case should be presented in detail for the purposes of study and comparison by the observers.

In resuming, the author says: "The biological alterations in protozoa of which the change of form is one of the most easily recognized are dependent on internal and external factors. It is to be supposed that certain changes in the exterior media, as, for example, the artificial culture media and also the products of syphilitic disease, may bring about marked changes in the form of the *spirochaeta pallida* which, indeed, shows that the body has a remarkable power of change, but does not justify the assumption that it has the power of forming a particular sort of an independent variety."

To prove whether certain varieties are transient in character or are independent and capable of being inherited, experimental methods of propagation are of more value than the observation of morphologic characteristics. Investigations in this line seem to show that all effort to change a "pure" race so that the new characteristics are hereditary are unavailing; that although great variations are seen a certain normal "mean" is retained. The author's observations on the spirochete tend toward the same result as regards the spirochete in syphilis, and he concludes that marked variability of the *Spirochaeta pallida* exists not only from a morphologic, but also from a biologic standpoint. In all probability, the spirochete is incapable of transmitting the new characteristics to descendants.

GURD, Ann Arbor, Mich.

A POLYNEURITIC SYNDROME RESEMBLING PELLAGRA-ACRODYNIA (?) SEEN IN VERY YOUNG CHILDREN. ALBERT H. BYFIELD, Am. J. Dis. Child. 20:5 (Nov.) 1920.

This is a report of seventeen cases of an epidemic erythema in children all of whom are under 4 years of age. The studies extend over a period of five years.

Clinically, these patients exhibited many interesting symptoms and signs. Predominant were the trophic changes found in all. There was alopecia, falling out of the teeth, reddening of the skin, neurokeratitis and changes in the nails. In the nervous system there were paresthesias of the hands and feet, diminished cutaneous sensibility in some, constant rubbing of the skin, pulling out of the hair, reflexes diminished or absent, muscular weakness and atrophy. Mentally the patients exhibited dulness and apathy alternating with restlessness and sleeplessness. In bed they assumed a fairly characteristic posture, lying on the side with the head buried in the pillows. They presented a picture of extreme wretchedness. The skin evidenced vasomotor disturbances in cyanosis and lowered temperature in the extremities. There was a nonconfluent erythematous rash recurring at intervals of about two weeks, which somewhat resembled pellagra. Desquamation was marked on the tips of the fingers and toes, and in some cases it was also seen on the thorax and upper arms where the lesion presented a morbiliform or bullous appearance. In the digestive tract, there was obstinate anorexia, but no vomiting or diarrhea; there was also some constipation. In the advanced cases there was unusual sweating. In the genito-urinary system, there was urinary frequency and often pyelitis. The laboratory examinations showed a consistent leukocytosis of 11,000 to 30,000 but no predominance of polymorphonuclears in the differential count. The stomach contents were invariably negative. The urine showed acetone, and, during the pyelitis, pus cells. The spinal fluids were negative with the exception of increased globulin. The Wassermann tests were negative.

The duration of the disease varied from two to eight months, and fourteen of the seventeen patients recovered.

A careful survey of the family histories and other facts failed to reveal any dietary deficiency; in fact, some of the patients were breast-fed at the time of the onset, and the mothers' diets were adequate. Many cases began as bad colds or influenza. Diphtheria-like organisms were found in many throat cultures but the patients gave a negative reaction to the Schick test, and the course of the disease was not altered by the administration of diphtheria antitoxin. No hereditary influences could be found. Seasonal distribution was the nine months between October and July. The fact that all presented leukocytosis argued for an infection—in fact, two cases cleared up after tonsillectomy and one after a nasal surgical operation.

The diagnostic possibilities were numerous. Pellagra was considered, but there was no sharp line of demarcation in the skin lesion; no common dietary error; no history of pellagra in the family; the age of the patients was uniform; there was insufficient digestive disturbance; lack of recurrences; frequent history of infection; occurrence in breast-fed babies; aggravation of symptoms in cold weather and prolonged duration of symptoms. Motor neuritis was ruled out because of the preponderance of sensory phenomena. Arsenic poisoning and ergotism were easily excluded. Trophoneuroses offered slight fundamental similarity, but differed as to age incidence. Acrodynia or "epidemic erythema" (Weston) is proposed, because of its close resemblance, but all reports of this disease show that the patients had conjunctivitis, spasticity and convulsions. The possibility of a postinfluenzal condition, such as a radiculitis or an infectious polyneuritis, is considered and supported by bibliographic reviews, but left merely as a consideration.

As for the treatment, gavage and forced feeding were found to produce the best results, and, under this routine, improvement began and continued favor-

ably in fourteen cases. Forced feeding was not attempted in the three fatal cases as they were early cases and the patients died in a short while. One patient died of pulmonary complications.

PATTEN, Philadelphia.

ERGEBNISSE DER BLUT UND SPINALFLUESSIGKEITS-UNTERSUCHUNG BEI LUETISCHEN HIRN UND RUECKENMARKS-ERKRANKUNGEN (RESULTS OF A STUDY OF THE BLOOD AND SPINAL FLUID IN SYPHILITIC DISEASE OF THE BRAIN AND SPINAL CORD). HERMAN KRUEGER, Monatschr. f. Neurol. u. Psych. 48:18 (July) 1920.

The value of the "four reactions" of Nonne—cell count, globulin, spinal fluid Wassermann and blood Wassermann—is beyond question. Nevertheless, practically all series of studies have shown that there are cases of central nervous system syphilis, proved clinically, therapeutically and pathologically, in which some or all four reactions are negative. As Rost has pointed out, negative reactions do not exclude syphilis; parenchymatous, endarteritic and even meningeal foci may exist without demonstrable fluid change. Even in general paresis, in which we are most likely to find all reactions positive, negative reactions may occur. These cases are sufficiently numerous to detract greatly from the diagnostic significance of the four reactions.

If all four reactions are positive, we are dealing with syphilis. It may involve the substance of the brain and cord, or it may be confined to the meninges (as is probably true in many of the secondary cases with positive fluids).

The author reports the results of the study of the four reactions in 310 cases of general paresis and taboparesis, eighty-four cases of tabes dorsalis and seventy-two cases of cerebrospinal syphilis.

General Paresis and Taboparesis.—The blood Wassermann reaction was positive in 96 per cent. of the cases; the spinal fluid Wassermann reaction in 93 per cent.; pleocytosis (over 10 cells) was present in 97 per cent., and globulin in 99 per cent. All reactions were positive in 88 per cent. of the cases. In no case were all reactions negative. In 12 per cent. of the cases, one or more reactions were negative. These negative reactions were not necessarily found in incipient cases, stationary cases, or cases in remissions. Many were typical cases, running a typical course, some of them rapidly progressive. In no case was it possible to render all the reactions negative by treatment.

Tabes Dorsalis.—The blood Wassermann reaction was positive in 85 per cent. of the cases; the spinal fluid Wassermann reaction in 77 per cent.; pleocytosis was present in 88 per cent.; globulin in 90 per cent. In 3.5 per cent. of the cases all reactions were negative. The findings were less constant than in paresis, and the effects of treatment more marked.

Cerebrospinal Syphilis (Meningitic, Gummatous and Vascular Forms).—The blood Wassermann reaction was positive in 79 per cent. of the cases; the spinal fluid Wassermann reaction in 66 per cent.; pleocytosis was present in 80 per cent., and globulin in 80 per cent. All reactions were positive in 51 per cent. All reactions were negative in 4 per cent. The effect of treatment on the reactions was greater in this group than in the other groups, and this may be responsible for some of the negative cases. It is not responsible for all of them.

Summary.—1. Negative findings do not exclude syphilis of the central nervous system.

2. With all four reactions positive, the disease with which we are dealing is in all likelihood syphilitic. The Wassermann reaction in the spinal fluid is specific, and if it is positive, it has the same significance as the four reactions in toto. Pleocytosis and a positive globulin reaction are not specific, and occur in numerous other diseases. In case of doubt, the effect of a course of treatment on the spinal fluid will decide.

3. The four reactions in themselves give little help in the differential diagnosis between the types of central nervous system syphilis. We can get some information by watching the results of treatment. Marked improvement in the fluid under treatment is strongly suggestive of cerebrospinal syphilis as differentiated from paresis and tabes.

4. Laboratory findings are valuable, but cannot replace clinical study. One cannot diagnose diseases of the brain and cord in the laboratory.

SELLING, Portland, Ore.

THE PATHOLOGY OF "SO-CALLED" LETHARGIC ENCEPHALITIS.

FELIX STERN, Arch. f. Psychiat. 61:621, 1920.

In a closely printed article of sixty-seven pages, the author first discusses the proper meaning of the term encephalitis and incidentally that of inflammation, and endeavors to limit the name to certain cases and classes of cases. He then discusses in great detail the histopathology of the four cases that he had examined, going superficially into the clinical histories. He attempts to classify his cases according to the findings in different stages of the disease and in typical and atypical cases, and finally he endeavors to establish a pathologic differential diagnostic syndrome of lethargic encephalitis or, as he prefers to call it, polio-encephalitis (Economo's type). He summarizes his own conclusions as to the histopathology in typical cases somewhat as follows: The picture is that of disease extending over large regions of the brain, but intensified by foci of interadventitial or peri-adventitial infiltration by lymphocytes and their derivatives, plasma cells and polyblasts, and accompanied by alterations in the ganglion cells, acute swelling and advanced degeneration, slighter alteration in the nerve fibrils and marked focal degeneration of the myelin sheaths, with active proliferative changes in the cellular glia and in later stages of the fibrous glia also, and with proliferation of the cellular elements of the vessel walls. The passage of hematogenous elements into the ectodermic tissue is found to a slight degree and some formation of new vessels is seen. Diffuse inflammation of the meninges always takes place, but there is no direct relation or subordination of the encephalitic process to the meningitic. In the brain the gray matter is the area of predilection, especially the thalamus and the central gray matter from the third to the fourth ventricle. Diffuse alterations in the greater part of the nervous tissue of the cortex with glia reactions, without any trace or with small sclerotic foci with reparatory proliferation of the glia and myelin sheath and sometimes slight diffuse gliosis in the area of earlier exudations, are characteristic of the disease. These are the typical accompaniments of the disease. The author looks on hemorrhages, which are usually slight, thrombosis, slight thrombotic softening and infiltration by polymorphonuclear leukocytes as accidental accompaniments, such as may be seen in many other conditions, and which are fre-

quently absent in encephalitis. Economo's opinion that the leukocytic infiltrations may be rapidly passing initial phenomena is probably correct, and the author calls attention to the fact that in epidemic meningitis also, the primary leukocytic foci are followed rapidly by plasma celled and lymphocytic infiltration. The free gliogenous or angiotic granular cells containing fat are more characteristic by their absence than by their presence in acute cases of lethargic encephalitis except in small thrombotic softenings. Although veins are more constant seats of infiltration than arteries, some arteries are infiltrated, and the predilection of the infiltration for veins is not characteristic of lethargic encephalitis alone as in epidemic cerebrospinal meningitis the veins are the preferred seat of infiltration in the pia and arachnoid.

The cells forming the infiltrative exudate are distinguished with accuracy as lymphocytes, plasma cells, polyblasts (cells with certain resemblances to plasma cells but which do not show the metachromatic staining and the clear "court" of the former cell; they are usually round or ovoid and the nucleus is much darker staining than that of the fibroblasts) and a cell with a round pale nucleus, originating in the vessel walls. The last cell is commonly called epithelioid, but the author objects strongly to the name and hopes it may shortly disappear as it is used indiscriminately for gliogenous elements for Maximov's polyblasts and for the large cells seen in tubercles. A negative characteristic of some value in the diagnosis of lethargic encephalitis is the small number or complete absence of granular (fat) cells, with the exception of those directly attached to the vessel walls.

The author's consideration of the etiology is based on a small number of observations made by his own countrymen, and he ignores the large amount of experimental and clinical observations made in other countries. He brings out rather clearly, however, the fact that there is more than one variety of encephalitis seen in epidemics of influenza.

He explains the predilection of the infiltrative process for the central gray matter of the pons, peduncles, etc., to the terminal character of the arteries of these areas, also to the increase of blood pressure in them caused by the arterial branches coming directly from the carotids, assuming as a primary fact that the blood vessels are the seat of the beginning of the toxic effects of the originator of the disease.

His atypical case was one in which there was a large red softening in the cortex caused by thrombosis of the pial veins, purulent meningitis, multiple hemorrhages and other conditions. Space is lacking for a detailed account of the case of the patient with an abscess in the right lung and pneumonic foci in the left lung, with many bronchiectases and emboli in one branch of the right pulmonary artery. On this case he attempts to base conclusions as to the origin of lethargic encephalitis as the case resembles one described in Economo's report and an organism was found, a "diplostreptococcus" similar to that found by Wiesner in Economo's case. He tries to explain the presence of a leukocytic infiltration and other conditions by the varying action of one and the same organism at different times under different conditions, giving as an example the action of *Spirochaeta pallida* in different forms of syphilis. The most valuable part of the article lies in the attempt to limit the term encephalitis to inflammatory processes and to the careful and detailed description of the histopathology of the four cases given.

The illustrations are not of the usual high standard to which we have been accustomed in German work.

GURD, Ann Arbor, Mich.

SYNDROME OCULO-SYMPATHIQUE DE CLAUDE BERNARD-HORNER PAR COMMOTION D'OBUS — PHENOMENE DE L'ADDUCTION OCULAIRE PROVOQUEE PAR TOUTE EXCITATION PERIPHERIQUE (CLAUDE BERNARD-HORNER OCULO-SYMPATHETIC SYNDROME AS A RESULT OF DETONATION SHOCK, WITH OCULAR ADDUCTION ON PERIPHERAL STIMULATION). A. LÉRI and J. THIERS, *Rev. neurol.* **26**:808 (Nov.) 1919.

In the case reported by the authors, there was a history of loss of consciousness, following shell explosion, on two occasions (July, 1916, and December, 1917) unaccompanied, on either occasion, by evidence of external injury. Following the first accident, persisting auditory defect, on the left, with perforation of the left tympanum, tinnitus, vertigo and headache was noted. Examination, subsequent to the second accident, revealed pronounced vertigo with uncertain, hesitating, lateropulsive gait and very pronounced Romberg symptoms. The left ear showed slight perforation of the tympanum with greatly diminished auditory acuity. The Valsalva sign was positive and the Weber test was localized to the left. The tendon reflexes were present and equal, and there was no clonus or Babinski sign. Sensation was undisturbed, and there was no sign of disorder of cerebellar function. The left pupil, however, was much smaller than the right although neither showed disturbance in reaction to light or accommodation. There was slight exophthalmos on the left, and the palpebral fissure on this side was narrower than on the right—in effect, the Claude Bernard-Horner oculopupillary syndrome. It is of interest to note, in this connection, that Cobb and Scarlett¹ report *enophthalmos* in each of the eleven cases of Claude Bernard-Horner syndrome comprising their series. It might be added that in Léri and Thiers' case, vision was apparently normal in both eyes and no disturbance was noted in ocular movement save that there seemed to be slight instability of the eyeballs in direct vision, and occasional nystagmoid movements were noted on the direction of the gaze laterally upward and to the right. The Wassermann reaction was negative, and there was no history or clinical evidence of syphilis.

On later examination, in connection with the Bárány test, it was noted that when cold was applied to either external auditory canal, there appeared, uniformly, a very definite syndrome characterized, objectively, by complete adduction of the left eyeball, contraction of the superficial muscles of the right cheek, and, finally, failure of equilibrium and falling. Subjectively, there were noted intense vertigo and diplopia. On further investigation, it was found that this syndrome could be produced by practically any type of peripheral stimulus, thus application of cold to the nostril or to the buccal mucosa, pinching of the skin, electrical stimulation, etc. In attempting an explanation of the mechanism underlying the production of this reaction, the authors direct attention to the possible causative relation of the existing labyrinthine defect, occurring as a result of the detonation-shock, citing, in support, a case in which there was observed a similar reaction, although much milder in type, and in which there was a definite history of labyrinth injury as a result of a fall from a horse.

RAPHAEL, Kalamazoo, Mich.

1. Cobb and Scarlett: *Arch. Neurol. & Psychiat.* **3**:636 (June) 1920.

SOME RECENT RESULTS IN THE INVESTIGATION OF THE
RELATIONS OF SPIROCHETES IN THE BRAIN IN GENERAL
PARALYSIS OF THE INSANE. F. JAHNEL, *Allg. Ztschr. f. Psychiat.*
75:503 (May) 1919.

The author reviews the work already done in investigation of the spirochetes' presence, distribution, etc., in general paralysis of the insane, and calls attention to the sources of failure always to find the organism, namely, their presence in swarms like bees in certain areas only, their selection of certain areas in a given case to the exclusion of other areas, and their periodic increase and decrease in number. The last cause is the expression of a biologic characteristic of all spirochetes that are active in inducing illness. The spirochetes do not entirely disappear at any given time, but become so reduced in number that great difficulty is experienced in isolating them. The author has established this phenomenon not only by clinical and microscopic observation, but also by experimenting with large amounts of blood injected at the time of the lessened number of spirochetes, and he concludes that spirochetes are present at all times in all cases of general paralysis of the insane although he may be able to show their presence in a part only of all cases. The author's present efforts are not toward obtaining spirochetes in all cases, but to determine the localization of the spirochetes in a large number of cases and the relations of the spirochetes to the tissues and the conditions under which the organisms arise and multiply in the nervous system.

The author's earlier works have identified two types of localization of the spirochete, in foci or disseminated. The present work adds a third localization, that of the vascular type in which the cortical vessels are specially the seat of the organisms. The spirochetes are not found in relation to all the vessels in a case, but usually all the vessels in a circumscribed area show the invasion of their walls by the organisms. Both veins and arteries show sheaths of the germs which also penetrate the vessel walls and lie in thick masses, sometimes lengthwise at other times crosswise. The parasites are frequently in such numbers that under a low power the vessels invaded by them appear darker than other vessels, and in oil immersions the vessels appear black, and it is only on using the fine micrometer that the individual spirochetes come to view. The cortical parenchyma is not entirely free from the spirochetes in these cases, but their number is extremely restricted and varies directly as the distance from the vessel wall increases. Sometimes the perivascular sheath is so excessive that the parasites invade the surrounding tissue and by the union of several of these small foci one of the large or "swarm of bees" type of focus results. On the edge of one of these giant foci the vascular type is frequently seen in its purest form.

The capillaries are invaded by the spirochetes in the same manner as the larger vessels. It is impossible to ascertain the relations of the spirochetes to the different vessel walls as the great number of the organisms prohibits close observation. Sometimes the spirochetes are in such numbers that the vessel appears like a tube composed entirely of spirochetes. Sometimes one portion of the spirochetes lies in the vessel lumen, at other times one portion is affixed to the vessel wall and the other portion lies in the parenchyma. At branching of the vessels the spirochetes frequently form a wedge or funnel passing from one vessel to the other. When marked infiltration of the larger veins by plasma cells and lymphocytes is seen, the spirochetes are seen between

these cells. The spirochetes pass through all the walls, even the intima is so invaded by them that it stands out as a blackish wall formed by concentric rings of spirochetes.

The vascular type shows certain interesting characteristics. Sometimes the spirochete sheath around a vessel ceases abruptly, sometimes gradually; at some divisions of the vessels many spirochetes are seen, at others few or none; there seems to be no regularity in these relations. Large veins often show a spirochete sheath passing only half or three-quarters way around its lumen, and the other half or quarter will show only scattered organisms.

An important point seems to be that when the vascular type is found well marked, it is seen in all portions of the brain and not only in a limited area. The white substance seems to be exempt from the spirochete as a rule, but in one case the author found spirochetes in the perivascular spaces in the white substance. The organisms are frequently found directly under the pia, in or under the glial limiting membrane. As a rule, the molecular layer is free from parasites, but sometimes when they are plentiful in the next layer a few wander into the molecular layer.

In one case, foci were found directly under the pia and in the same case "bee swarm" foci and also disseminated spirochetes were found in the ganglion cell layers. In one case spirochetes were seen forcing their way into the pia. This is probably a rare occurrence.

Two clinical histories are related in detail with resultant necropsies, both showing the vascular type of spirochete localization, but the clinical courses of the two cases and the degree of invasion and degeneration of the parenchyma were so utterly dissimilar in the two cases that no possible conclusions could be drawn either as to symptomatology or as to the severity of the process in cases of vascular localization.

The question arises whether the vascular type of localization may not be an intermediate term of a later "bee swarm" foci arrangement, and one is led again to a consideration of the path by which the spirochete enters the brain. Spirochetes have been seen in the blood, but may it not be that the lymph stream alone carries them to the brain, which the marked vascular localization would render probable, and their escape again into the blood stream would explain their presence there? All these interesting details and questions presented by the author must incite to a more general and thorough study of the whole question into which light can only be brought by large numbers of detailed observations. The author's figures are extremely conservative in regard to the percentage of cases in which he has found spirochetes. Taking a series of unselected cases, he found spirochetes in the dark field in 50 per cent. and in cut sections in only 25 per cent.

GURD, Ann Arbor, Mich.

DU TRAITEMENT DE LA PARALYSIE GENERALE PAR L'INOCULATION DE CERTAINES MALADIES FEBRILES (PALUDISME — FIEVRE RECURRENTE) (TREATMENT OF GENERAL PARALYSIS BY INOCULATION OF CERTAIN FEVERS — MALARIA, RELAPSING FEVER). P. PAGNIEZ, *Presse méd.* **28**:736 (Oct. 16) 1920.

This paper is chiefly a review of German work published since 1918, particularly that of Mühlens, Weygandt and Kirschbaum, "Die Behandlung der Paralyse mit Malaria und Rekurrenzfieber," *München. med. Wchnschr.* **67**: 831 (July 16) 1920. The results obtained by these inoculations were not spec-

tacular, but seemed sufficiently encouraging to warrant study and further experimentation.

Among the different fever-provoking agencies tried, tertian plasmodia came to be preferred. From six to eight paroxysms (sixteen in one case) were induced in a course before it was terminated by quinin medication. The maximum body temperature reached was 106.8 F., compared with a maximum of 105.4 and an average of 104 degrees for relapsing fever. The mechanism of the curative process was not clear. Some experiments on animals suggested that the spirocheticidal action of high temperature might alone be responsible for whatever improvement was effected.

Of 33 patients treated, 3 died of intercurrent diseases, 1 died of a complication dependent on the therapy, 12 had treatment discontinued for various reasons, 5 were not favorably affected, while 12 were benefited to such an extent that the improved status could fairly be termed a remission. Some of these parietic patients recovered sufficiently to return to responsible positions, and there carry on their former occupations satisfactorily. Most of these remissions had lasted for several months, and in one case for as long as three years. They exceeded the usual expectation of remissions under other forms of treatment. The serology was variously influenced. Generally blood Wassermann reactions were unchanged, while spinal fluid reactions showed more or less improvement.

In selecting patients suitable for this treatment, it was necessary to exclude all old weakened persons, all cases of long standing, and patients whose quinin tolerance did not show a sufficient margin of safety for the after-treatment.

HUDDLESON, New York.

THE THERAPY OF GENERAL PARALYSIS. R. WEICHRODT, Arch. f. Psychiat. 61:132, 1920.

This article contains a critical review of the treatment of general paralysis. The author includes in detail the results of his own researches with therapeutic measures. His conclusions, stated in the introductory paragraph, are that "The therapy of general paralysis is as yet an unsolved problem; the progress of the disease continues and leads with few exceptions to death in from three to five years." With due regard for the frequent occurrences of remissions and the rare cases in which remissions have lasted for years, he comments "theoretically one may rightly believe that such improvements may become permanent and as Spielmayer has stated, from the pathological-anatomical viewpoint there is nothing that excludes the possibility of recovery. Of course, it could only be recovery with defect, but the observations of remissions even in advanced stages show that the brain even in diffuse processes has great possibility of compensation." Remissions in general paralysis stand in close relation to the biologic characteristics of the spirochetes. He refers to the work of Jahnel, which seems to show that as the paralytic attacks are explainable by flare-ups in the increase of spirochetes which after the attack die rapidly, so the remissions may be due to periods in which most of the spirochetes have died, and the few that remain are in an inactive phase. One may only expect therapeutic success by reaching the spirochetes, and only when their increase can be prevented can there be a remission or can the disease be brought to a standstill.

The author presents a chronologic and systematic consideration of the therapeutic efforts that have been reported in the literature. His discussion

of the progress of arsphenamin therapy covers fully the more important contributions dealing with methods and their application. From the experience of the clinic at Frankfort he concludes that there have been no serious bad effects from its use. Now and then there have been improvements, but it is questionable whether these can rightly be ascribed to the therapy. Following a review of the therapeutic effects of mercury, iodine preparations and collargol, he gives in detail some personal researches with methylene-blue and trypan-blue.

He believes that when one summarizes the results of experiences with pharmacologic agents, it is evident that no one of these is able to produce a permanent improvement in the disease. The remainder of his contribution is devoted to a discussion of the therapeutic influence of febrile temperatures on the course of mental disorders and specifically on general paralysis. A review of the extensive literature dealing with this subject forces the conclusion that febrile disease does not always influence a psychosis, but the undoubted improvements and long remissions that have been observed justify a further study of the influence of temperature on general paralysis. He records his own researches in this direction in much detail. He produced febrile temperature with inoculations of the blood of the tertian type of malaria in two cases of well advanced general paralysis. Both patients developed malaria with high temperature, but the course of the general paralysis was uninfluenced. Two other patients in whom paralysis were less advanced improved so much that they were able to leave the hospital; they said that they felt well and that they noticed an improvement of their memory. Too much weight should not be placed on these results as the experiments are recent, but they justify further investigations with this method. As to whether the influence excited by febrile conditions comes from the high temperature or from the hyperleukocytosis, a review of the literature and his own experiments indicate that it is not the result of the increase of leukocytes. From observations showing that intercurrent infectious diseases have a favorable influence on an existing syphilis, the author, working with Jahnel, was able to produce death of the spirochetes in rabbits with syphilitic chancre by exposing the animals to a temperature of from 42 to 43 C. at three intervals of time. This suggested a line of experiments to produce high temperatures in the body of patients with general paralysis. The only way of producing sufficiently high temperature in the body of man is the hot bath and the question arises whether by this means an influence could be exerted to check the growth of spirochetes in the brain. This method of attacking the problem seems hopeful enough to warrant further experiments.

BARRETT, Ann Arbor, Mich.

XANTHOCHROMIA WITH REPORT OF THREE CASES. LOUIS A. LEVISON, *Arch. Int. Med.* 26:4 (Oct. 15) 1920.

Xanthochromia is not an uncommon finding in spinal fluids, but a true Froin's syndrome is much less frequent. Yellow fluids occur in many conditions, including general infections, icteroid states and obstructive lesions of the central nervous system. The color is not in itself pathologic, but the coagulability of the albumin content is and does not occur except in lesions in which the circulation of spinal fluid is cut off. Froin's theory as to the color is that it is obtained from bilirubin, but others do not sustain this theory and suggest that it originates from hemorrhage, although no explanation is offered for the

absence of albumin. The important findings then, in a yellow spinal fluid are, increase in the number of cells and the tendency to coagulation. Yellow fluids, however, occur sometimes when no cause can be found.

The author reports three cases of yellow fluids bearing out the preceding discussion. The first case is that of an inoperable intramedullary spinal tumor. The spinal fluid was a rich canary yellow in color; it had 22 cells per c.mm., 18 of which were lymphocytes; an excessive amount of globulin, with partial spontaneous clotting; and negative Wassermann and Fehling's reactions. The second case was diagnosed as syphilitic pachymeningitis with gummatous formations. The spinal fluid was a bright canary yellow, it contained a large amount of globulin which did not coagulate, 78 to 140 lymphocytes per c.mm. and a strongly positive Wassermann reaction. The fluid became clear after a few months of active antisymphilitic treatment. The third case was an example of a yellow fluid in a man with biliary cirrhosis. The spinal fluid was under tension; it was a dull brownish yellow color and showed an increase in globulin but no coagulability; the cell count was 18 cells per c.mm., all small lymphocytes, and the Wassermann and Fehling's reactions were negative.

The author concludes that Froin's syndrome is diagnostic of an organic spinal lesion of the obstructive type, but a mere color change is not important.

PATTEN, Philadelphia.

PSYCHIC DISORDERS IN TABES. KASIMIR BRODNIOWICZ, *Allg. Ztschr. f. Psychiat.* **75**:701 (Aug. 14) 1919.

While tabes and paresis are commonly associated, there are many cases of tabes accompanied by psychic disturbances that differ from those of paresis in their character and the course of the disorder. The author reviews the literature dealing with psychoses in tabes in which various opinions are expressed as to the development and course of tabes psychoses. It has been held by some that in a reflex way the various neurologic disturbances of tabes may, by influencing the brain centers, bring about the occurrence of a psychosis. Others have asserted that tabes acts only as a predisposing factor for the development of elementary psychic symptoms which may be further elaborated into a psychosis. Anatomic studies have definitely shown that many tabes psychoses are to be considered as part of a general syphilitic or metasyphilitic disorder, which may be an arrested paresis or a mild cerebral syphilis.

The author reports three cases that he had observed in the Rostock Psychiatric Clinic. The first of these was that of a man, 46 years old, who had a syphilitic infection at the age of 19. Tabetic symptoms appeared ten years later. When admitted to the clinic, his pupils were myotic and stiff. The knee and Achilles' reflexes were absent. There were Romberg's sign, marked hypotonia and sensibility disturbances. The Wassermann reaction of the blood was positive. Psychotic symptoms appeared soon after his infection. For a considerable time these were little more than a marked irritability and defective judgment. There later developed a paranoid attitude toward his wife with delusions of suspicions. This subsequently was extended to include others who came in contact with him, and finally led to delusions of persecution which he continued to hold without deterioration of memory or judgment for thirteen years. The second case was that of a man aged 32, who had slowly coursing tabetic symptoms; stiff pupils, disturbances of sensibility and gastric crises. Somewhat atypical were the presence of a Babinski reflex on the left side and

a negative Wassermann reaction of the blood and spinal fluid. There was, however, a slight pleocytosis. At the age of 40 a mild depression developed with an attempt at suicide. In the further progress of the disorder there were delusions of poisoning, states of excitement and delusions of jealousy. His interest in his work fell off, and when released, he was unable to support himself. The third case was that of a man, 52 years of age, who on admission was in the phase of development or remission of a mild tabes. His pupils were narrow and had a slowed light reaction. There was a slight weakness of one facial nerve. The knee jerks were diminished and the Achilles' reflexes were lost. The Wassermann reactions of the blood and spinal fluid were negative. The psychotic features appeared at the age of 47, when he had two transitory attacks of excitement with much unclearness and a subsequent amnesia. It resembled the acute delirious states occurring in syphilis, that have been described by Plaut and others.

The author concludes that the tabes psychosis may occur in two types: one, an acute, transitory crisis-like disorder, and the other, as a more chronic, paranoid psychosis.

BARRETT, Ann Arbor, Mich.

LA NEVRITE HYPERTROPHIQUE DE L'ADULTE (HYPERTROPHIC NEURITIS IN THE ADULT). M. DIDE and R. COURJON, *Rev. neurol.* **26:825** (Nov.) 1919.

On the basis of six personal cases and a careful study of the literature, the authors believe that the essential clinical picture, in this condition, may be described as follows:

The characteristic muscular atrophy seems to begin, consistently, in the upper extremities (very exceptionally the atrophy may begin in the lower extremities), and with very slow progression proceeds centrad, along the limb, from the periphery, until, finally, the entire segment may be involved. Signs of the tabetic series (ataxia, loss of sense of motion and position, hypotonus, etc.) are practically absent, and signs of the cerebellar series (dysmetria, intention tremor, asynergy, etc.) are but exceptionally observed and only in the very advanced stages. In connection with the latter, the authors question whether, in these cases, such reaction may not be wholly explainable on the basis of asynergy between unequally damaged muscular units rather than on actual cerebellar disturbance. The tendon reflexes remain intact for a long time, but, finally, may become diminished or even abolished. The idiomuscular reflexes follow the curve of the atrophy, appearing, at first, very active and later falling below the norm. No disturbance is noted in the cutaneous or ocular reflexes, nor is there evidence of nystagmus, strabismus, or exophthalmos. The electrical reaction depends entirely on the state of the muscles; but, in any case, it seems that reaction of degeneration appears only in the very late stages. Objective sensibility, as a rule, remains normal although a certain degree of hypesthesia and agnosia have been noted. The nerves involved are frequently painful to pressure, and dull pain and tingling are spontaneously noted along their course. Palpation reveals definite nerve hypertrophies which may be either diffuse or nodal in type. No disturbance is observed in genital function, sense organs or mentality.

In discussing the etiology of the condition, Dide and Courjon emphasize the fact that, from their study, there seems to be no hereditary or familial relationship nor any special predilection for age or sex. They believe that

neither syphilis nor leprosy are of any etiologic importance and suggest the possibility of some, as yet, undetermined infection with predilection for neuritic site.

Nosologically, the authors suggest the following classification:

1. Juvenile type, such as of Déjerine-Sottas and Marie-Boveri, which may be complicated by symptoms of the tabetic or cerebellar series.
2. Adult type, which may be of two kinds, namely, (a) simple type (as described above), and (b) complex type (rare)—simple type complicated by symptoms of the cerebellar series.

RAPHAEL, Kalamazoo, Mich.

INVESTIGATIONS ON THE CAPACITY FOR PSYCHIC FUNCTIONING OF THOSE HAVING HEAD AND BRAIN INJURIES, BY KRAEPELIN'S METHOD OF CONTINUOUS ADDITIONS. G. Vos, *Allg. Ztschr. f. Psychiat.* **75**:265 (April) 1919.

In this article is reported experimental work showing the capacity for psychic performance of a group of patients with head injuries, and following the progress of the disorders in their effects on psychic functioning. The author employed Kraepelin's method of continuous additions. This method offered possibilities of obtaining data for comparison of performance curves of those with brain injuries showing symptoms of more or less organic defects, with head injuries of the traumatic neurosis type.

The subject was required to add combinations of two numbers of one digit for a period of fifteen minutes. A rest period of fifteen minutes was followed by another fifteen-minute period of addition; a rest of ten minutes was then followed by a final addition period of fifteen minutes. The progress of the performance was divided among five minute phases, and the results were arranged in graphic curves. The author studied seventy-five cases of head injuries. In many of these the experiment was repeated at intervals of from four to six months. Of twenty-two subjects who had repeated the test, fourteen showed a greater or less improvement in their performance; in six it was unchanged, and in two the ability fell off.

These three groups of improved, unchanged and failing performance in repetition of the experiment all showed a striking agreement in their method of work, that is, the form of the performance curve. This seemed to eliminate any possibility of wilful alteration of the results in the anticipation of compensation for their injuries. The results of these experiments justified the following conclusions: 1. The greater number of those injured in the head or brain showed an improvement in their performance. A few showed a regression of performance capacity. 2. The results did not always agree with the progress of the physical symptoms of the subject. On the one hand, it was found that severe physical symptoms went hand in hand with good psychic performance, while on the other hand, with slight physical involvement psychic performance was poor. Improvement in the physical sphere did not always cause an accompanying betterment of performance ability. 3. The performance curve of patients with head and brain injuries showed marked individual differences. By repetitions of the experiment, the subjects held to a fixed type of performance, so that later curves were almost photographic reproductions of the earlier ones. 4. Most of the subjects showed at the beginning of the test an inhibition so that the performance of the first five minute phase was less than that of the second. 5. Most subjects had little ability to benefit by practice. Where this was marked it was usually associated with marked

fatigability. 6. The total performance of those with head or brain injuries was low, being about one third of that of normal subjects of the same intellectual level. 7. The larger number of cases of his series could be divided between two groups: (1) patients with marked organic disturbance and, (2) patients whose symptoms were largely functional (psychogenic). Between these two groups were many transitions so that a sharp separation was not possible. 8. The calculation ability of Group 1 was markedly better than that of Group 2. Poorest were the performances of a group of patients with traumatic neuroses of other origin, that had been studied for comparison. 9. Psychic effort in cases of head injuries may precipitate a convulsive attack. Therefore caution should be used in deciding as to the working capacity of those who have had head or brain injuries, especially in respect to those who have had epileptic attacks following the injury.

BARRETT. Ann Arbor, Mich.

LA LIPODYSTROPHIE PROGRESSIVE (PROGRESSIVE LIPODYSTROPHY). L. BOISSONNAS, *Rev. neurol.* **26**:721 (Oct.) 1919.

On the basis of twenty-two cases (two personal), the author concludes that the clinical picture in this disturbance may be thus standardized:

There is, fundamentally, a very gradual atrophy of the adipose tissue of the upper portion of the body, attended by fatty hypertrophy below the waist. The atrophy is first noted about the face, following which the neck, thorax, shoulders, arms and abdomen are successively involved. The mammary glands, however, are, as a rule, not involved. The skin itself shows no definite changes although there have been noted increased sudomotor activity and a peculiarly malodorous sebaceous secretion. In regard to the concomitant hypertrophy of the lower extremities, it has been found that the gluteal and gluteocrural areas are first involved, and in fact, may be the only areas affected, although in most cases this process later affects the thighs and legs, generally stopping at the ankles. The fat is deposited in a uniform manner, following limb contour, although occasionally, one limb may appear, as a whole, more hypertrophied than the other. The muscular and osseous systems show no disturbance. Sensation remains essentially unchanged, nor is there definite evidence of characteristic mental change. For the most part, cranial and peripheral nerve function remain undisturbed, although, in isolated cases, mention is made of exaggerated tendon reflexes, unilateral tremor, abolition of plantar reflexes, diminished pupillary reaction to light, nystagmus, facial tics and dermatographia of the lower extremities. Boissonnas finds no evidence of relation to age or sex, but notes that this disease has been observed following infectious diseases and the establishment of menstrual flow. In discussing the etiology, the author feels that the essential cause is some, at present, more or less obscure lesion of the central nervous system and not, as some have suggested, primary endocrine dysfunction.

RAPHAEL. Kalamazoo, Mich.

NEOLOGISMS OF THE MENTALLY DISORDERED. S. GALANT, *Arch. f. Psychiat.* **61**:12, 1919.

This is a psychopathologic study of the peculiarities of speech so frequent among certain types of the mentally disordered. The few studies previously made on this subject have concerned the neologisms of dementia praecox. In the present contribution the author has studied not only those of that disease, but those of epilepsy, in much detail. His purpose was to find the ideas

that were back of the neologisms or neologism complexes and the origin of the peculiarly formed words. He believed that the neologisms did not appear suddenly, but had passed through phases of development, and that their content must represent ideas that had been much in the patient's mind in health or in the early years of his disease. His researches convinced him that most of the neologisms are not entirely senseless; that they are often ideas over which the patient has long brooded, and which are with a certain logical method expressed automatically in the form of neologisms. He attempts to demonstrate that there are types of neologisms characteristic of the different forms of dementia praecox and in this respect the form of neologism may be of diagnostic value. In the paranoid type the neologism occurs as a part of a system. It is intimately connected with the personality of the patient. His proposition is supported by a detailed analysis of the productions and life histories of two patients. The chief difference between the paranoid and catatonic type of neologism lies in the precipitating stimulus. In the paranoid type the stimulus that leads to the neologism is purely psychic and the neologisms are psychogenic. Those of the catatonic come from many stimuli; these are not psychogenic, but are from motor speech elements. In the paranoid type it is easy to follow the gradual development of the neologisms, but not so in the catatonic. The catatonic patient is not conscious that he uses new words; they come from the tongue and not the brain. Neologisms rarely occur in the hebephrenic type. These the author designates as symbolic. They are words of ordinary speech used symbolically and are not true neologisms. Although the foregoing three types of neologisms are well differentiated from one another, there are cases in which the types intermix or occur as transition forms.

Next to dementia praecox, neologisms are most frequently found in epilepsy. In outward form the neologisms of the two diseases are difficult to distinguish. The newly formed words of the epileptic patient are speech contaminations, abnormal combinations and peculiar usages of suffixes; while these occur in dementia praecox, there are differences in that the epileptic does not create new words except when they are in continuity with other words he is speaking. They cannot be understood except when interpreted in connection with the psychic peculiarities of the epileptic. They are determined in part by the impairment of memory, the circumstantiality, the tendency to form rhymes and to speak in symbols present in this disorder. The author presents a number of extensive thought productions of epileptic patients that are analyzed in much detail.

The neologisms occurring in other psychoses show nothing characteristic.

BARRETT, Ann Arbor, Mich.

THE POSITIVE WASSERMANN REACTION IN NONSYPHILITIC DISEASES OF THE BRAIN. FELIX STERN, *Arch. f. Psychiat.* **61**:725, 1920.

Plaut, Jahnel, Hauptmann, Mucha, Kramer and Zadek are quoted as having observed a positive Wassermann reaction in the cerebrospinal fluid in tuberculous and other meningitides not syphilitic. Plaut asserts that many of these cases occurred in syphilitic patients in whom the meningitis caused an undue permeability of the meninges and allowed the passage of the reagent from the blood into the spinal fluid, but Stern claims that too many cases have been reported in which the blood was negative and the spinal fluid positive

to permit much weight to be placed on Plaut's explanation of the phenomena and considers, therefore, that every unspecific reaction should be carefully noted. The cause of this unspecific reaction has been assumed by some observers to be the cell degeneration of the pleocytes and by others to be the alteration in lipid substances, but no definite conclusion has yet been reached. The author urges a more critical study of all the reactions of both blood and fluid, and after noting that many observers have found a positive blood Wassermann reaction with negative fluid in nonsyphilitic tumors, he reports his own case of a combination of malaria with a small sarcoma of the cerebello-pontile angle in which the Wassermann reaction of the blood was weakly positive and of the fluid +++ positive from 0.1 on.

An interesting point in the case was that treatment by quinin and such drugs always caused such great improvement of the ordinary tumor symptoms, such as vertigo, vomiting and headache, that it was only on the appearance of marked vestibular disturbance with deafness that tumor was suspected. Decompression was then practiced, and a radical operation was in preparation when the patient died. The tumor was the size of a small pigeon's egg. There was no leptomeningitis and no vessel alteration. The malaria was tertian in character, and the plasmodia were found constantly in the attacks which were repeated several times with a lapse of from three to nine months between the exacerbations. The diagnosis of a nonsyphilitic cause for the fluid Wassermann reaction was made first from the absolutely negative history of the patient, then from the failure of all syphilitic symptoms and finally from the peculiar reactions of the fluid itself, in which there was a marked increase in albumin with complete absence of pleocytosis, the paradoxical phenomenon found in different affections leading to increased pressure in the brain, but not found in syphilitic affections. A positive Wassermann reaction in the blood serum in malaria had been observed many times before the war, and the experiences of the war have shown that it is relatively frequent.

Stern considers the positive Wassermann reaction in the fluid in his case as "specific" for malaria, just as in syphilis it is "specific" for that disease, i. e., the reaction stood in direct causal relation to the malaria as it was not to be explained either by pleocytosis, which did not exist, or by reduction of lipid substances as the tumor was very small and showed absolutely no degenerative changes. In all probability, the meninges were rendered more permeable by the action of the malaria permitting the passing of the complement reagent into the fluid from the blood. Whether the complement may be formed in the meninges is still unknown.

GURD, Ann Arbor, Mich.

THE CAUSES OF FEEBLEMINDEDNESS IN EARLY LIFE. SCHOTT, Arch. f. Psychiat. 61:195, 1919.

This is a study of such causative factors of feeble-mindedness as might be ascertained from the histories of 1,100 patients as given by their families. Appreciating the defects that are inevitable in such information, the author made a critical study on the occurrence of the following factors: (1) hereditary stigmatization including alcoholism of the parents and ancestors, (2) blood relationship and illegitimacy, (3) head and birth injuries, (4) brain disease and convulsions, (5) infectious disease, (6) rachitis and tuberculosis, (7) psychic traumas of the mother during pregnancy.

As regards the age from which the mental deficiency dates, he found that 60 per cent. of feeble-mindedness may be considered congenital and 18 per

cent. as acquired during the first years of life. The remaining 22 per cent. resulted from factors active during the second to the fifth year, and a few developed in later years.

His conclusions are: As a general cause, diseases of the brain are of the highest importance in the production of feeble-mindedness. These occur in at least one third of the series. Heredity and alcoholism of the ancestors is the sole cause in one third of the cases. In about one seventh of all cases no cause can be found. The remaining, about one third, are ascribable to a variety of causal relationships.

The most important medical problem in this field is to investigate and attack all pathologic processes that may act injuriously on the child's brain, either intra-uterine or after birth, particularly the infectious diseases. The campaign against feeble-mindedness is intimately connected with the problems of hygienic and social betterment.

As special causes, hereditary stigmatization is of the greatest importance. In more than two thirds of these cases a moderate degree of feeble-mindedness was present in the ancestors. Blood relationship and illegitimacy are not important causes. Head injury is of importance only when there are symptoms indicating severe injury, such as unconsciousness, vomiting and spasms. Birth injuries are in a similar class. Diseases of the brain are of much significance, and prevention of these will have an influence in reducing the number of feeble-minded. Convulsions, which occur in about 8 per cent. as the sole cause, are to be regarded as due to brain disease. The conquest of the infectious diseases and the elimination of their causes will have a beneficial influence. Whatever can be done toward diminishing the amount of tuberculosis and rachitis will be of great value.

As there are in about one third of all cases several causal relationships, it becomes essential to direct the attack on feeble-mindedness from various aspects.

BARRETT, Ann Arbor, Mich.

EIN WEG DAS WESEN DER "GENUINEN EPILEPSIE" ZU
ERFORSCHEN (A METHOD OF STUDYING THE NATURE OF
GENUINE EPILEPSY). PROFESSOR HAUPTMANN, *Monatschr. f. Psychiat.*
u. Neurol. 48:1 (July) 1920.

The author points out the serious limitations of our knowledge of genuine epilepsy. In searching for the cause we have to consider three factors: hereditary factors, metabolic disturbances and histopathologic changes.

The question of hereditary factors can be solved only by careful anatomic studies. These must include not only such gross changes as the heterotopias, but also the most minute developmental anomalies. If these studies are carried sufficiently far, he believes that no epileptic brain will be found entirely normal. Probably many nonepileptic brains will show similar pictures. Assuming such congenital anomalies as one essential, how can we explain the development of the disease with its convulsive seizures and psychic equivalents? The author believes that a second fundamental factor comes into play—a metabolic fault.

If we assume that faulty metabolism is essential to the development of epilepsy, we must determine whether this fault is primary, or, in its turn, secondary to degeneration of brain tissue; or whether epilepsy is the result of both factors, coexistent but independent. It is the possibility of a metabolic disturbance secondary to brain damage or brain anomaly that makes it difficult to determine the initial factor.

The author believes that we may reach a solution of the problem by a study of war material. Here we can rule out hereditary factors, at least in a large percentage of cases. We can study in such cases (1) what metabolic disturbances result from cranial lesions in general; (2) what metabolic disturbances result from convulsive seizures per se (to be worked out in jacksonian cases in which the seizures are of local origin); (3) what metabolic disturbances arise in the true post-traumatic epilepsies, that is, in those cases, not to be explained by local irritation, in which the attacks develop long after healing of the wound. We must determine what takes place during this latent period; and we must determine whether changes are occurring in the brain which render it more susceptible to irritation, or whether metabolic disturbances are developing which must reach a certain degree before they cause epileptic symptoms.

Hauptmann summarizes the possibilities with respect to the pathogenesis of genuine epilepsy as follows:

1. Congenitally abnormal brain—exogenic irritants.
2. Pathologic metabolic products—endogenic irritants. (a) Normal metabolic products. (b) Pathologic metabolic products. (1) Primary metabolic disturbance. (2) Secondary metabolic disturbance dependent on cerebral degeneration.
3. Normal brain—metabolic disturbance.
4. Primary, progressive cerebral disturbance.

As the author suggests, the problem is a large one. Just what phases of metabolism are to be studied he leaves to the discretion of those who undertake the work.

SELLING, Portland, Ore.

POST DIPHTHERITIC OCULAR PARALYSES WITH REPORT OF THREE CASES. M. LOMBARDO, *Am. J. Ophth.* **3**:747 (Oct.) 1920.

The author reviews the literature using an intensive bibliography. He reports three cases, the first of bilateral isolated paresis of accommodation and the second and third of isolated paresis of the right external rectus. In these cases the paresis manifested itself from four to seven weeks after the appearance of the primary disease. With the view of determining whether the usual treatment is of any avail, either to shorten the duration or in retarding the final result of the paralysis, no treatment was advised. The paralysis lasted from four to six weeks, and at the present time the condition of the muscles is entirely normal.

Remarks: These three cases and many others reported in the literature show that the paralysis of the eye muscles may be a sequel of diphtheria irrespective of the use of antitoxin and the age of the patient. Diphtheria may paralyze the eye muscles without producing paralyzes of muscles of other organs. Postdiphtheritic paralyzes of eye muscles pass away spontaneously.

Treatment has no influence on the course of these paralyzes. In the writer's cases paralysis lasted from four to six weeks. This is the usual duration of post diphtheritic paralysis, as far as we know from the cases reported in the literature.

The eye muscles in postdiphtheritic paralysis usually regain their function entirely.

REESE, *Philadelphia.

MICROGYRIA AND ITS EFFECTS ON OTHER PARTS OF THE
CENTRAL NERVOUS SYSTEM. W. PAGE MAY, *Brain* **43**:26 (May)
1920.

A brief case report is given with a rather detailed discussion of pathologic findings. The reader is referred to the original for the details.

The patient had a left hemiplegia in early childhood, which persisted to the time of his death at the age of 36. He showed mental deficiency which increased as he grew older, also epileptiform convulsions, which were likewise progressive.

The main pathology was arrest of development of certain parts of the entire central nervous system, which caused secondary and tertiary atrophy and resultant striking organic and functional changes. The right hemisphere was one third smaller than the left, the pyramidal tract was atrophied, the left cerebellar hemisphere and left half of the spinal cord were distinctly wasted. The cortex on the right showed shrunken convolutions and a decidedly lessened proportion between the gray and white matter. The basal nuclei were similarly affected, showing paucity of fibers and cells. The left hemisphere was normal in every way, and, contrary to expectation, showed no evidence of compensatory hypertrophy.

The author defends the name microgyria as truly representative of the condition, stating that the name does not involve the causal factor, yet indicates clearly the seat of the grossest change. The case differs from those reported by Mott and Tredgold in that their cases were either definitely cortical or basal, whereas this case falls into an intermediate grouping, being partly cortical and partly basal.

PATTEN, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY AND SECTION ON NEUROLOGY

Joint Meeting, Nov. 9, 1920

WALTER TIMME, M.D., *President*

POLIOMYELITIS. Presented by DR. ISADOR ABRAHAMSON.

Dr. Abrahamson called attention to the existence, almost in epidemic intensity, of a disease syndrome transitional in symptoms between lethargic encephalitis and poliomyelitis. He discussed the difficulties of diagnosis and the variability of the clinical manifestations of lethargic encephalitis. Lethargic encephalitis having died away until only chronic and remitting cases persisted, poliomyelitis had reappeared in epidemic form in Boston and subsequently in New York City. In the latter city, the board of health had already reported ninety-three cases, most of which occurred in October.

Undoubtedly, many of the cases in this epidemic of poliomyelitis showed a clear cut poliomyelitis syndrome. But besides such irrefutable cases, there were numerous others characterized by a common syndrome, which supplied, as it were, the missing link between the average syndrome of lethargic encephalitis and of poliomyelitis. These transitional or polio-encephalitic types were both mild and severe. Patients with mild cases showed nasal and pharyngeal catarrh with fever, malaise and implication of the facial muscles, sometimes with diplopia. The following are brief notes on cases of the severe type:

First Case.—A boy of 16 had pharyngitis, nausea and vomiting followed by diplopia. The same evening left facial palsy developed, followed by involvement of the left side of the tongue. The temperature rose rapidly. The next day, there was oculomotor involvement, paralysis of the left side of the face and tongue and of the motor fifth nerve; there were left palatal palsy and marked midbrain tremor of the upper extremities. The abdominal reflexes and the right knee jerk and both Achilles' reflexes were absent, the left knee jerk was diminished. There were no Babinski reflex or Kernig's sign, and no retraction of the head. The spinal fluid showed 66 lymphocytes. The diagnosis was bulbar and midbrain poliomyelitis. The patient died that night.

Second Case.—This patient was a boy of 9, with early diplopia and rigidity of the neck. There was right dropped foot; a left Babinski reflex and Kernig's sign were present. The patient was lethargic for forty-eight hours. The spinal fluid within forty-eight hours of the onset showed 149 cells, 90 lymphocytes and excessive albumin and globulin; Fehling's reduction. The patient recovered with the exception of dropped foot on the right.

Third Case.—A 2 year old child, whose mother had lethargic encephalitis with considerable residues, showed a poliomyelitis syndrome.

Fourth Case.—Four members of the same family had acute sore throat just before a fifth member, a boy of 15, showed diplopia, ptosis on the left, tremor of the extended right hand, paralysis, and later atrophy of the left upper extremity, weakness of the right side of the face and retraction of the head.

There were no evidences of Kernig's or Babinski's signs, no nerve tenderness or hyperesthesia. The patient's condition improved, with the exception of the paralyzed left upper extremity.

Fifth Case.—A woman of 39 was aphasic for four days. She lost power first in the right upper extremity and then in the right lower extremity. There was myoclonic twitching in the upper right side of the abdomen. The left pupil was larger than the right, rigid and ovoid. Seventh nerve weakness, left externus and internus, right externus and flaccid weakness of the left upper extremity, bilateral Babinski sign and marked midbrain tremor of the left upper extremity were present. The blood pressure, heart vessels, and urine were normal. The Wassermann test was negative. The spinal fluid showed old blood (from seven to ten days old) and 14 cells, all lymphocytes.

Dr. Abrahamson said that he had been in communication with Dr. Zingher of the health department, who had studied the spinal fluids in the reported cases of poliomyelitis of this epidemic, and he said that the vast majority showed a great excess of lymphocytes, excess of globulin and Fehling's reduction. Almost all had at the outset a catarrhal inflammation of the nose and throat and a few had gastro-intestinal signs. The same peculiarity was noted in lethargic and influenzal encephalitis.

DISCUSSION

DR. M. NEUSTAEDTER said that the cell count, globulin and albumin content of the spinal fluid alone without the clinical findings were no index of any particular disease. In any inflammatory condition of the meninges, irrespective of the etiology, there are alterations in the spinal fluid, in its cytology as well as its chemistry. Dr. Neustaedter said that his experience and the literature showed that early in poliomyelitis there was marked lymphocytosis and that later polymorphonuclears predominated. Reports of other investigators showed that with the Lange test about 45 per cent. of the cases of poliomyelitis gave the syphilitic curve. In his work on complement fixation of spinal fluids with poliomyelitis antigen he had obtained some gratifying results in poliomyelitis and recently also in lethargic encephalitis. Dr. Neustaedter thought that this specific complement fixation test should be tried more extensively and that there might be a specific reaction pointing more definitely to the etiology of the two diseases. He stated that he had conducted neutralization tests with poliomyelitis virus and the serum of recovered lethargic encephalitis patients since last March. This work had not yet been reported. The results showed that there was a close point of contact between poliomyelitis and lethargic encephalitis.

DR. DAVID J. KALISKI (by invitation) said that he was not convinced of the value of the complement fixation test for poliomyelitis, using the antigen perfected by Dr. Neustaedter. Most of the serums sent by Dr. Neustaedter were negative, while a few of the controls (not cases of poliomyelitis; most of them old specific cases or other neurologic conditions) gave fixation with the antigen. Therefore the question of the specificity of the antigen was to be considered. Using the organisms of lethargic encephalitis isolated by Loewe and Strauss as antigen in a complement-fixation test for this disease, he had not been able to decide definitely as to the value of the reaction, principally because of a falling off in the cases of the disease when he was prepared to study the problem. The work was proceeding and would be reported later.

Dr. Kaliski stated that there were no definite diagnostic criteria in the cerebrospinal fluid which served to distinguish poliomyelitis, lethargic encephalitis and the polio-encephalitis types. While there was likely to be a larger number of cells in the fluid in the former condition, with increased globulin reaction, and even faint opalescence of the fluid due to a large number of cells, this was not diagnostic, but merely suggestive. Hemorrhage and even xanthochromia had been seen in a few cases of lethargic encephalitis. The curves obtained with the colloidal gold test were not characteristic of poliomyelitis. A rare case had been reported showing a curve similar to that of paralytic dementia or syphilis, but he had not been able to confirm this in any of his cases. In encephalitis likewise no characteristic or constant deviations from the normal were found. In a few cases of superior poliomyelo-encephalitis, no changes with the colloidal gold test were found, while the lymphocytes were usually only moderately increased, rarely above 50 to 100 cells to the millimeter.

Poliomyelitis, encephalitis, poliomyelo-encephalitis and tuberculous meningitis frequently gave similar findings in the fluid as regards the cells, the globulin and the presence of sugar reducing substances, and the deviations were usually in degree only. Of course, in the last disease (tuberculous meningitis) the finding of tubercle bacilli cleared up the diagnosis. From the standpoint of the pathologist, the diagnosis of any of these conditions with certainty was impossible, with the exception of tuberculous meningitis.

Dr. J. H. LEINER spoke of four patients at the Lebanon Hospital on the service of Dr. William Leszynsky, in three of whom there were facial palsy and meningeal irritation that later proved to be of poliomyelitic origin. One child was somnolent and had no Babinski or Kernig's sign; knee jerks were absent. The patient died within forty-eight hours. One child, with facial involvement, gave a history of vomiting and gastro-intestinal disturbance three or four days before coming to the clinic. The illness then assumed a poliomyelitic character.

Dr. ABRAHAMSON expressed the hope that by calling attention to the current epidemic, the symptoms and signs of which seemed to fall between the known varieties of poliomyelitis and encephalitis, new data might be collected and the relationship between influenza, lethargic encephalitis and poliomyelitis might be established.

PUPIL DILATATION AND THE SENSORY PATHWAYS. Presented by Dr. JOSEPH BYRNE.

Dr. Byrne considered the mechanism of pupil dilatation as set down in the textbooks unsatisfactory. Experimenting with cats, Dr. Byrne found that after section of any considerable portion of the afferent pathways, pupil inequality ensued. Thus after section of one sciatic nerve or of the lumbosacral nerve roots, the contralateral was smaller than the homolateral pupil, whereas after section of the brachial plexus, or of any of the cervical nerve roots, as well as after hemitranssection of the spinal cord in the upper or lower cervical region, or in the lower thoracic or upper lumbar region, the homolateral pupil was smaller. From eight to twelve days after section of one sciatic nerve (or other of the enumerated operations), intravenous injection of epinephrin caused the relatively constricted pupil to become, for a

time, larger than its fellow. This phenomenon is known as paradoxical pupil dilatation and Dr. Byrne's experiments were the first in which paradoxical effects have been observed after lesions of the afferent pathways. All previous observations had been made after lesions of the efferent pathways, that is, of the cervical sympathetic nerve. Besides anatomic section, Dr. Byrne had injected alcoholic solutions into the sciatic nerve and into the nerve trunks of the brachial plexus. The results corresponded closely with those obtained after anatomic section.

From the effects of complete cord transection at various levels above and below the upper thoracic region (inferior ciliospinal center of Budge) on the pupillary phenomena noted after section of the afferent paths, it was concluded that in the normal waking state afferent impulses from all parts of the periphery are constantly impinging on the cells of the lateral gray column in the upper thoracic region on both sides. These impulses determine the efferent flow along the cervical sympathetic nerve which keeps the pupil dilated. There is, however, another factor in pupil dilatation that depends on inhibition of the sphincter muscle. But this forms the subject of a separate study. Dr. Byrne's experiments give no support to the assumption of a hypothetic cerebrospinal dilator pathway running from the region of the pons to the lower ciliospinal center. In no instance had Dr. Byrne been able to obtain effects on the pupil after hemitransection of the cord in the cervical region which could not as readily be obtained after posterior nerve root section. However, hemitransection of the cord at about the level of the second cervical root caused marked homolateral pupil constriction. This, in Dr. Byrne's opinion, was in part the result of injury of the root pathways of the fifth cranial nerve and not the effect of lesion of any special cerebrospinal dilator pathway. Dr. Byrne thought that his experiments opened the way for a more exact interpretation of pupil inequality as encountered clinically, for example, in tabes, etc., whereas they seriously brought into question the value of the so-called Klumpke-Déjerine sign, which was usually interpreted to mean injury of the efferent cervical sympathetic pathways.

DISCUSSION

DR. SHERWIN (by invitation) asked for information as to the structures on which epinephrin was supposed to act in eliciting paradoxical pupil dilatation.

DR. K. SELLARDS KENNARD (by invitation) called attention to the fact that pupil inequality was a somewhat common occurrence among cats and asked whether any steps had been taken by Dr. Byrne to eliminate possible error from this source.

DR. WALTER MAX KRAUS asked whether histologic studies had been made on the cells of the lateral gray column and on the dorsal nuclear column. It seemed as if the pathways involved in the pupillary phenomena were identical with the pain paths. The main afferent sympathetic paths were those of pain which probably crossed in the spinal cord after entering with the sympathetic nerve.

DR. THOMAS HAYES CURTIN (by invitation) asked how the epinephrin was administered and what its effect was on the musculature. He spoke of various methods of producing dilatation. It might be produced by the effect of the administration rather than by the drug itself.

DR. BYRNE, in closing, said that the site of action of epinephrin in eliciting paradoxical phenomena was presumably the myoneural junctions in the iris. As in the case of the blood vessels, epinephrin had a two-fold action on the pupil, namely, a primary dilating action, followed by a secondary constricting action.

Replying to Dr. Kennard's question, Dr. Byrne said that in all cases careful preliminary tests had been made, and after such tests many animals had to be rejected. Replying to Dr. Kraus, Dr. Byrne said that the histologic studies had been made and marked changes had been observed, for example, after sciatic section, in the cells of the dorsal nuclear as well as in the lateral gray columns. The changes were evident on both sides but were more marked on the contralateral side in the case of the lateral gray columns (Budge's inferior ciliospinal center). Dr. Byrne agreed with Dr. Kraus that the paths involved in the pupillary phenomena were closely related to, if not identical with, the pain paths. The experimental studies were undertaken in the first instance with the hope of substantiating physiologically and anatomically Dr. Byrne's hypothesis as to the mechanism of pain.

A STUDY OF MOTOR APHASIA OF THE CORTICAL OR MIXED TYPE. Presented by DR. SAMUEL BROCK.

An American railroad mechanic, 40 years of age, had used alcohol to excess, formerly with frequent intoxication. Cigaretts were also used to excess, but no drugs. The family history was unimportant; there was no history of head injury; venereal infection was denied.

While in France in army service about two and a half years ago, the patient suddenly fell forward unconscious. On recovering, speech was affected and the right arm and the right side of the face were paralyzed. The face and arm had practically recovered, but the speech defect had improved little. Since the onset he had had seizures of a jacksonian type, beginning in the right side of the face and involving the right arm and leg. Examination revealed motor aphasia. The gesticulations, mimicry and slight irascibility seen in this type of speech difficulty were manifest. Stereotyped phrases were used, such as "just the same," "sometimes pretty good, sometimes I could do nothing," "If I come here, I'll have to give it up," "if you can," "Boston," "I cannot move." He was able to count up to seven and to speak the first two letters of the alphabet.

When asked to write his age he wrote 1881, the year of his birth. His replies were not the exact answer to the question asked, but closely related to it; for example, "when" seemed to be mistaken for "where." He was able to write few words from dictation, and could not write simple sentences or numbers of more than four figures. He appreciated his inability and noticed his mistakes. He was able to write his name. Copying printed or written words was done quite well. Reading printed or written words was accomplished slowly but correctly. He recognized all objects, and gave no evidence of apraxia.

There was no gross impairment of motor power. No Babinski or allied reflexes could be elicited. They were all exaggerated and equal on both sides, except that the right Achilles' reflex was slightly more active than the left, and at times the right knee jerk predominated. In the sensory sphere, an area of hypesthesia was noted, together with analgesia on the anterior and posterior portion of the right side of the chest and adjacent neck, down to the

level of the abdomen. There were no ataxia or adiadokokinesis and no Gordon-Holmes phenomenon. Tremor of both hands was present.

He had suffered from dyspnea on exertion, cardiac palpitation and precordial pain. The evidence of cardiovascular disturbance and the sudden onset postulated a vascular accident in a branch of the left middle cerebral artery supplying the posterior part of the left inferior frontal convolution and adjacent motor area. The residuum was considered to be either a cyst or an old area of softening. An operation was proposed with the understanding that although there was only a slight possibility of improving his condition, there was a possibility.

OPERATIONS AND FINDINGS OF ABOVE DESCRIBED CASE. PRESENTED
BY DR. K. WINFIELD NEY

Under local anesthesia, a large flap was turned down in the left parietal region. About 7 cm. above the external auditory meatus a large cyst was found. Vessels were ligated around the cyst. During the ligation a large amount of fluid escaped from the cyst, after which it collapsed, leaving a cavity about 2 cm. wide and 1 cm. deep. The thin upper wall of the cyst was resected leaving a cavity that had the characteristic yellow appearance of degenerated brain tissue. The lesion had been located accurately.

About a year ago, seventy or more craniotomies performed under local anesthesia were reported before the Society by Dr. Ney, and since that time he had added about twenty-five additional cases. He stated that he was becoming more confident of the value of this procedure. The majority of neurologists were somewhat skeptical in regard to intracranial operations because of the high mortality. In this series of operations under local anesthesia there have been no operative deaths. The local anesthesia never had to be supplemented by a general anesthetic, nor had it been necessary to discontinue any operation because of pain, shock or any of the disturbing factors of general anesthesia. Blood pressure seldom changed during the operation. There was no pain unless the dura was pulled or too greatly traumatized. The vascular instability associated with many brain operations, such as edema, etc., had not been a complication following invasions under local anesthesia. The infiltration of the tissues gave an almost bloodless scalp incision which was also helpful in the elimination of shock. The prospects of intracranial surgery under this form of anesthesia, Dr. Ney felt, were most encouraging.

DISCUSSION

DR. NEUSTAEDTER asked concerning the results of the operation. Had the jacksonian attacks been affected?

DR. TIMME asked whether any nystagmus had been present.

DR. BYRNE said that Dr. Taylor had been using local anesthesia at Fordham, and that he personally could not speak too highly of its advantages. The neurologist, incidentally, was given an opportunity to test cortex sensibility.

DR. BROCK said that the patient was no better. The jacksonian seizures were just as frequent as before, and the aphasia was the same. There was no nystagmus before or after operation. The findings of this case bore out earlier studies on aphasia. A cortical lesion at the entering and leaving pathways, caused a mixed aphasia, which was of distinct value from a localizing standpoint.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

*Regular Monthly Meeting, Nov. 18, 1920**EVERETT FLOOD, President, in the Chair*

ORGANIZATION AND FUNCTIONS OF THE NEUROLOGIC DEPARTMENT OF THE MASSACHUSETTS GENERAL HOSPITAL. DR. E. W. TAYLOR.

Dr. Taylor spoke of their successful organization based on an "integrating policy." The principal factors were a larger personnel, special assignments for individual research and for intensive clinical work in a limited field, intimate association with other departments of the hospital and close alliance with the medical school of Harvard University. Mention was made of the importance of the social service department.

A final necessity of a well-organized clinic is a clinic secretary whose duty it is to see that the clinic runs smoothly and effectively, that the patients are attended to and that the needs of the physicians are met.

RESIDUAL SPINAL CORD SYMPTOMS FOLLOWING INTRASPINAL ANESTHESIA. DR. W. E. PAUL.

Dr. Paul reported two cases of loss of sphincteric control immediately following intraspinal anesthesia. In both at least partial anesthesia of the saddle back area existed.

The first patient was a single man, 50 years of age, operated on for fistula in ano in September, 1913. He passed his urine the first day after operation, but the second day and for fifteen months thereafter a catheter was required. When the dressings were removed he was found to have fecal incontinence. There was a typical saddle back area of anesthesia except that the skin of the penis was not involved and the scrotum was less anesthetic than the peri-anal and posterior thigh regions. The blood Wassermann reaction was negative. Knee jerks were present but the ankle jerks were not obtained. The pupils were equal and reacted normally. In August, 1915, the bowels were constipated and a laxative was required, but care was needed to prevent accidents. The patient felt his sphincter was improving a good deal. After fifteen months a catheter was used only once a day and after the twentieth month only three times. He noted, however, some escape of urine at times and on passing urine the amount was small and the urine would start and stop. The anesthesia persisted over the saddle back area twenty-three months after it first developed. For a year he had little, if any, sexual feeling, but after a year there was some return with good erections.

The second patient was a married man, 45 years old, operated on in the middle of June, 1920, for double inguinal hernia. He had retention after the operation and required a catheter. Constant drainage was resorted to for a time, and he left the hospital at the end of three weeks. Possibly he had some trouble in controlling gas immediately after the operation, but there was no incontinence of the bowels. About two months after the operation he had involuntary urination, especially at night. Four months after the operation he was using a catheter twice daily but passed considerable urine voluntarily with effort and straining. He had urgent desire to urinate when urine accumulated but after the catheterization was comfortable for about three hours. He

had involuntary passage of urine every night. The blood Wassermann reaction was reported as negative. The knee and ankle jerks were normally present. The abdominal reflexes were lively, but the cremasteric reflex was absent. The skin around the anus was anesthetic in moderate degree, but the scrotal and penile skin was not. The sexual function was preserved in good measure.

Dr. Paul refrained from attempting to state what the lesion might be. It did not seem likely that the needle or trocar caused the lesion as the symmetrical distribution of the anesthesia would not be likely to be due to direct trauma from the needle.

DISCUSSION

DR. GEORGE G. SMITH stated that he considered spinal anesthesia very valuable. It can be used when ether will almost certainly be fatal. The occasional bad results should be balanced against the probably fatal results from ether. In urologic work, spinal anesthesia is particularly useful; it is practical in cases of retention from stricture, especially when there has been an overdistended bladder for a long time and the kidney function is below normal. In such cases ether would almost certainly be fatal, yet with spinal anesthesia the stricture can be cut and the patient can begin immediately to drink water. It is difficult to explain such results as Dr. Paul has reported. The needle entering a vein and causing hemorrhage might have something to do with it. Such instances as the use of too hot a solution of an anesthetic may be passed over. It is possible for those things to happen but not probable. As to the toxic effect of the drug used, one might like to know what drug was used. Procain is preferable, and in a 2.5 solution, that is, a 5 per cent. solution diluted with an equal amount of spinal fluid, would undoubtedly have no toxic effect on the nerve substance itself. It is possible that tropacocain, which was formerly used, would be a little more toxic. It seems unlikely that there could have been complete sensory paralysis since, if there had been, the patient would have had no sensation from his bladder as it became filled.

A NOTE ON CERTAIN ELEMENTARY VISUAL HALLUCINATIONS IN THE PRESLEEP STATE. DR. SIDNEY LORD.

The word elementary is perhaps not happily chosen in designating the varieties of sense experiences in question, for it seems to be used in different ways; for instance, in specifying the origin of the experience in the sense organ itself in conditions of clouded consciousness, or as denoting the fact that such experience is elementary in duration and constancy, insignificant in influencing behavior or in causing any falsity of belief; or elementary in elaboration or completeness, that is, elementary in the sense of being unorganized rather than organized.

The phenomena in mind are elementary as concerns resultant behavior or deception of the intelligence, but are not truly elementary in the physiologic sense; that is, they are elaborated sensory images and are called hypnagogic hallucinations, hypnagogic illusions, perception phantasms or visions. Though they are sufficiently well known to receive the colloquial name of "faces in the dark," they are perhaps never entirely normal and, if almost invariably benign in prognosis, have not always been regarded as free from possible sinister import. There is not a large recent literature. These phantasms are not therefore frequent problems for the clinician. Occasionally, however, they are so often repeated or accompanied by such perturbation of mind as to

significance and course, occur with such vivid objectivity, and, at the moment, a fear so harassing that they compel more than the usual scant attention accorded them by the consultant. Moreover, when these phenomena are observed critically, the conviction is forced on one that they differ in various ways. There seems to be no doubt that some are pathologic and that some are not.

CASE 1.—A woman of about 30 was in a state of profound nervous exhaustion. Her illusions at bedtime were elaborate landscapes. There was no doubt in her mind as to their unreality, but there was a general anxiety about the presence of these illusions.

CASE 2.—A woman of 80, of alert and remarkable mind, but highly neuro-pathic, had from time to time been seeing ugly faces when the eyes were closed at night. No especial emotion accompanied these, or at least none was mentioned.

CASE 3.—An Armenian, about 50, presented himself complaining of insomnia and haunting visions at bedtime. He was intelligent, quite sane, sad, and much disturbed by the presence of these phantasms, profoundly so at the moment of their appearance, asking himself, "My God, why has this evil thing come to me? I am not crazy. What is it?" He described what he called "bad heads," "never a good head," and said, "The mouth keeps opening." He said the picture was always of a man or an animal picture. Asked what kind of an animal, he answered, "Never any kind that I saw before," except that the vision was, rarely, that of a serpent. This production of an image of a partly imaginary type is rather unusual. At least some of the images were gross exaggerations of anything in experience. He usually saw only the heads and not the other parts of the animals and men. They were not stationary; they always moved in one direction. The vision disappeared when he opened his eyes, to reappear on again closing the lids, but in "always different" guise. Sometimes it came when his eyes were open and it vanished when he shut them. He saw it moving away, and though he knew it was unreal, he was afraid; he pulled up the coverlet to try to hide from the horrid thing, closing his eyes to blot it out. It was grotesque, mysterious, unnatural; he was "awful afraid" but never deceived in his estimate of it. He tortured himself with the query, "Why does nothing beautiful ever come to me like this?" He never saw these things in the day, and never in the middle of the night; always at the first coming on of slumber. Graphically he stated that they were like moving pictures—never any noise. Asked why they come, he said, "I don't know why they come; they come themselves." Again, "Sometimes I watch it and then anything I remember comes." This last was a slightly different state suggesting the childish power of voluntarily evoking phantasms.

CASE 4.—A man of 50, intelligent, sane, neuropathic, had been subject for years to a variety of sensory shocks in the predormitium, to use Weir Mitchell's phrase. The visual images were usually seen at times of overwork and exhaustion. One type was the grotesque face which appeared with opening and shutting mouth. It was described as an unpleasant experience, accompanied by a general feeling of mental distress. Opening of the eyes dispelled the vision, but it tended to recur once or twice before he went to sleep. This was the common type. A much less frequent occurrence in this person was that of an extraordinarily vivid visual hallucination of scenery, of landscape. This occurred just before going to sleep when the senses were off their guard, but with the fully retained power of opening the eyes instantly to dispel the vision, which began with rudimentary, indistinct outlines of an object. Strikingly

like a developing photographic plate this vision expanded rapidly in detail and assumed the aspect of a landscape, surpassingly vivid and complete, indescribably clear and actual in appearance. The perception seemed to be one of transcendent brilliancy, vividness and beauty, quite beyond the realm of experience. It therefore seemed unnatural and awful. Fear arose and seemed to become overwhelming as the picture acquired a quality of being too real to be bearable. The patient felt as though he could put out his hands and pull out of the picture the objects which he saw. This was all so manifestly incongruous and false as to arouse the fear of something wrong and sinister—and the vision was then dispelled voluntarily by opening the eyes.

Such sensory impressions are different from dreamstuff. More nearly allied to dreams seems to be the vaguer imagery arising under similar conditions without any accompanying feelings of inappropriateness or dread of the unknown. The typical dream image is accepted by the subject without definite protest, so to speak, whereas these vivid phantasms are recognized as such and not accepted. Freud affirms, in speaking of hypnagogic hallucinations in general, their continuity with the dreams of the true sleep following. Dr. Lord is inclined to doubt this in relation to the more vivid and fear-compelling illusions spoken of.

These illusions are reported in part to emphasize two things which are not urged in the descriptions so far noted, namely, the quality of ultrareal objectivity felt by the perceiver and the degree of alarm experienced at the moment of illusion.

DISCUSSION

DR. A. W. STEARNS spoke of the relation of the fears to the illusions. He believed that the emotion was primary, and advanced the argument that the person being afraid had the illusions rather than that the person having the illusions became afraid.

DR. LORD stated that he had described the sensation of fear as it appeared to the person who had the emotions. To him there was a sequence in time. He saw the rudimentary vision begin without any emotional accompaniment. As it grew it was so unreal and extraordinary that it seemed to him to signify something sinister even though he recognized at the moment that it was a phantasm; hence, as he thought, the fear.

DR. E. W. TAYLOR mentioned a case of hallucination in an hysterical young man. He had the following experience: He was aroused by hearing a sound as of knocking immediately followed by a violent explosion. This was so vivid that he arose, turned on the light, went to the hall door and looked out expecting to smell smoke. He found nothing and returned to bed. This experience was repeated three times, the explosion occurring in different parts of the room accompanied by vivid red flashes of light. The realism continued so that each time he felt confident of the actual explosion in spite of his previous experience. This same night, while the lights were out, he had the illusion that the furniture was being violently moved; this illusion was immediately allayed by turning on the light. He had not before, and has not since, had similar hallucinations.

SOME OBSERVATIONS ON THE EYE IN DISEASES OF THE NERVOUS SYSTEM. DR. C. A. McDONALD.

Eight cases were reported. Three patients had double choked disk. One of the cases was diagnosed as a cardiovascular renal condition. There was an unusual degree of choking for this condition, but a low functional kidney

test corroborated the diagnosis. Another patient with a milder degree of choked disk and cardiovascular renal condition had a trace of albumin; the blood pressure was systolic, 230, diastolic, 140, and the retinal vessels were tortuous. The blood and spinal fluid Wassermann reactions were positive and the spinal fluid pressure was 350. A third patient in this group had double choked disk with a spinal fluid pressure of 90 and central chorioretinitis and numerous vitreous opacities, but the Wassermann reaction of the blood and spinal fluid was negative under potassium iodid and mercury treatment. The patient has improved much, but still receives mercury treatment.

There were three cases of unilateral choking of the disk, two following injury to that side of the head. The other, in addition to the choking of the disk, showed chorioretinitis in the vascular region and spots on Descemet's membrane. The importance of an examination of the media was emphasized.

There was one patient with optic atrophy with negative blood and a positive fluid, who improved considerably after receiving intravenous arsphenamin treatments. Another patient had complete optic atrophy of the left eye, failing vision (20/40) and a pale nerve head of four weeks' duration on the right. The blood and spinal fluid were positive for syphilis. After five intravenous arsphenamin treatments given weekly, vision was 5/200.

EXPERIENCE WITH SPINAL CORD TUMORS DURING THE PAST TEN YEARS. DRs. GEORGE CLYMER, W. J. MIXTER and HUGO MELLA.

DR. GEORGE CLYMER reported a group of cases occurring at the Massachusetts General Hospital since 1912, with nontraumatic lesions involving the spinal cord on which laminectomy had been performed or considered. In all of them the outstanding clinical picture was that of cord compression, with or without nerve root irritation.

The group consisted of fifty-two patients of whom thirty-seven were operated on; a lesion accounting for the symptoms was found in twenty-nine. One of the cases operated on with no findings at necropsy showed a tumor of the cord at a level slightly higher than that at which it had been localized. In four patients a lesion not found at first was found at a second operation. The final diagnoses in the remaining seven, in whom no lesion was found at operation, were: multiple sclerosis, myelitis, question of brain tumor, sciatica, cord tumor, question of syringomyelia and lateral sclerosis. Of the twenty-nine patients in whom a lesion was found, four were operated on twice, the lesion not being found until the second operation, or if demonstrated at the first operation, an attempt to remove it was not made until the second operation.

The symptomatology of the patients as they appeared in the hospital was: pain in thirty-two cases, numbness of legs in twenty-one, inability to walk or loss of strength or stiffness of legs in twenty-one, unsteady gait and ataxia in four, dragging of feet in three, limping in two, incontinence of urine in eight, incontinence of feces in eight (these two symptoms did not always occur in the same patient), urinary delay in two, constipation directly associated with the onset of other symptoms in two, constriction band occurring in three and spasmodic twitching of legs in one. These symptoms are not separated according to type of lesion because they seemed to occur rather indifferently in all types.

The age at the onset of the symptoms varied from 3 to 58, the average being 40. The duration of the symptoms prior to operation varied from three months to twelve years, the average being between two and three years.

The final results of operations are not at present available for the whole series. In eight cases, the lesion was removed at operation. Twelve patients had been relieved when discharged; fifteen were unrelieved and two died.

Diagnostically, pain is the most frequent symptom in lesions causing cord compression, but is not universal even in cord tumors. The next important symptom is a sensory level, but this also does not always occur. The greatest help in the diagnosis of these lesions has been given recently by Dr. J. B. Ayer in his use of combined cistern and lumbar puncture. (The foregoing is the outline of a preliminary report.)

DR. W. J. MIXTER spoke on surgical difficulties and described the technic of operation. The operative mortality should not be excessive but shock is to be expected. In a series of forty laminectomies he had had but one death, and that was due to operative shock. Another death, however, was that of a woman with malignant disease of the spine. A specimen was removed for diagnosis and a laminectomy was not performed. Death followed shortly.

The prognosis of spinal cord tumors depends on the type of tumor. Cases of intramedullary tumors have all resulted unfavorably. In only one case could it be removed, and then no improvement followed. Another type in which the prognosis is poor is metastatic malignant disease of the spine. Operation is justified for relief of pain, but the relief lasts only for a few months. Tumors arising from nerve roots and tumors of the dura respond to operative treatment. Another important factor is the position of the tumor in the canal. Those situated anterior to the cord are more difficult to remove and the danger to the cord is much greater. Tumors of the cauda equina can be treated more satisfactorily. Tumors of the dorsal region are the easiest to remove, those in the lumbar region come next and the cervical tumors are most difficult. The most recent advance in technic is the examination of the spinal fluid by combined cistern and lumbar puncture. This gives definite and positive information as to the presence or absence of block in the canal. It is of great importance in eliminating certain of the degenerative processes involving the spinal cord. Cistern puncture is a dangerous procedure in untrained hands. It should never be performed except by a person skilled in the procedure. This procedure is a definite advance, but should be used sparingly and with the greatest possible care and precaution.

DR. HUGO MELLA stated that in the cases reported by Drs. Mixter and Clymer a definite pathologic diagnosis was made in twenty-nine. The types varied greatly in their order of frequency. There were found recurrent metastatic carcinoma, fibrosarcoma, tuberculosis of cervical spine, endothelioma, neurofibroma, glioma, myeloma, dermoid cyst including cholesteatoma, cyst of cauda, enchondroma, adamantinoma, malignant leiomyoma, solitary tubercle and chondroma. Both intramedullary and extramedullary carcinoma were found. Sarcoma arose from either the vertebrae or meninges and usually invaded the cord substance. Kyphosis following tuberculosis of the cervical spine caused a "kink" in the cord which, if not corrected, may cause complete destruction of the cord. The solitary tubercle is difficult to locate in life and may break down causing transverse myelitis since they are usually intramedullary. Attention was called to the work of Mallory on the so-called endotheliomas. In his article in the *Journal of Medical Research*, March, 1920, he showed that the cells from which these tumors grow are a part of the arachnoid and should therefore be named arachnoid fibroblastoma as they "invade" the dura secondarily. Closely allied to this group are neurofibroma, fibroma molluscum or von Recklinghausen's disease, which may involve the nerve roots or the

cauda. The perineurium, according to Mallory, is probably not an extension of the arachnoid along the nerves, but an analogous differentiation of the mesenchyma around them and, if we accept these findings, should be named perineural fibroblastoma. Dermoid cyst, including cholesteatoma, does harm by causing cord compression. Adamantinoma was found in the region of the cauda. It is a rare tumor developing from remnants of the enamel organ and is usually found in the lower jaw. The cyst of the cauda was probably the result of an incomplete closure of the neural canal, the remaining sack being filled with fluid; these tumors are probably related to hydromyelia. These cases did not include those of trauma, gumma or abscess.

In general, the outstanding features of these cases from the pathologic standpoint is their remarkable diversity.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Nov. 18, 1920

DR. G. B. HASSIN, *President, in the Chair*

REPORT OF TWO CASES OF ENCEPHALOMYELITIS, WITH PRE-DOMINATING CORD SYMPTOMS. DR. PETER BASSE. (This paper will appear in full in the ARCHIVES.)

CASE 1.—A Chinese laborer, 50 years old, had urinary retention and weakness of all four extremities. The paralysis was not complete and was combined with marked ataxia. Patellar and ankle clonus was present and there was considerable muscular rigidity, especially in the trunk. Sensation was normal; the spinal fluid gave a cell count of 15 and weakly positive Ross-Jones and Lange reactions. After an illness of a few months the patient made a rapid recovery. The case was tentatively considered one of epidemic encephalomyelitis, with marked but temporary cord changes.

CASE 2.—A woman, 45 years old, was taken ill in August, 1919, with sore throat and nose bleed, followed by stiffness and pain in the legs. Diplopia was present for a few days, speech was slightly bulbar, and mental dulness and confusion were pronounced throughout the course. After a few weeks a picture of transverse myelitis in the thoracic region was established. At first the leg reflexes were increased, but they soon disappeared entirely. The spinal fluid gave a cell count of 26, a weakly positive Ross-Jones reaction and a fairly strong Lange reaction. Urinary retention and bedsores dominated the picture toward the end. The patient died on January 25, 1920.

The brain showed no gross changes and no important microscopic ones. The spinal cord was so extremely softened in the upper thoracic region that smears were stained in order to demonstrate that no pus was present, but merely necrotic cord substance. Numerous Gitter cells and fat globules were seen, but no micro-organisms. Sections from the rest of the cord showed extensive destruction of the white fiber tract and abundant fat granule cells. The ganglion cells were remarkably well preserved. Degeneration of white matter to the degree of necrosis was the main feature, inflammatory changes being less prominent. The case resembled others reported as instances of post-influenzal myelitis by Harbitz, in Norway, and others.

DISCUSSION

PROF. ANTON J. CARLSON: What was the condition of the sphincters toward the end? A considerable part of the sacral cord was intact.

DR. BASSOE: Yes, and in spite of that there was complete retention of the urine all the time. I don't remember about the anal sphincter.

DR. GEORGE B. HASSIN: In reference to the second case designated by Dr. Bassoe as myelitis, it would be more proper to call the condition of the spinal cord myelomalacia, or softening of the spinal cord. Just as softening of the brain occurs from vascular disturbances, a similar condition may obtain in the spinal cord from thrombosis or embolism, the former being much more frequent. The question is whether we can call such a condition myelitis. The myelomalacia in Dr. Bassoe's case involved exclusively the dorsal region, while the cervical, lumbar and especially the sacral, regions merely showed signs of secondary degeneration—with the hematoxylin stain. Dr. Hassin was sure that more refined staining methods will show even in these regions some definite changes.

In reference to the first case of lethargic encephalitis, he would like to ask Dr. Bassoe whether he remembered the condition of the pupils, because there is no lethargic encephalitis without some changes in the cranial nerves, especially the third, sixth and sometimes the seventh. In all of his nine cases of epidemic encephalitis studied pathologically, there were found most typical changes in the midbrain. It has been pointed out also in the ophthalmologic literature that some ocular changes are always present in this disease.

The spinal cord may be affected in epidemic encephalitis, but it never shows foci of softening, or necrosis which, if present, denote myelomalacia, as shown in Dr. Bassoe's second case. In epidemic encephalitis the inflammatory phenomena are infiltrative, in myelomalacia the changes could be termed alterative. Dr. Bassoe's remark that his first case was a most peculiar type of myelitis is practically correct. It is a peculiar type in the sense that the main changes are confined, not to the vessels as in epidemic encephalitis, but to the nerve elements themselves.

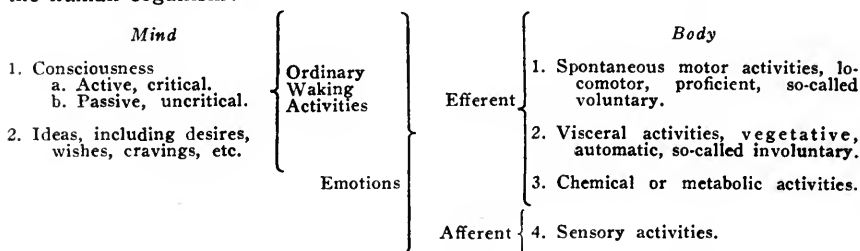
DR. BASSOE: In regard to the pupils in the first case, the case of the Chinaman, they were unequal at first, and then became equal, and they reacted normally at all times when I examined him. I did not see much of him during the first four or five days, when he was in the surgical department, but when I first saw him the pupils were equal. There were no cranial nerve changes that I could make out.

REPORT OF AN EPIDEMIC OF MENINGOCOCCIC MENINGITIS.

DR. RALPH P. TRUITT.

THE PRECISE LIMITATIONS OF THE INFLUENCE OF THE MIND UPON THE BODY. DR. MEYER SOLOMON.

The following schema gives a working classification of activities within the human organism:



Under the term "mind" I include the activities of consciousness, whether active or passive, and of ideas, including under the latter term wishes, cravings and desires as well as ideas.

Under the term "body" I include the activities of a spontaneous motor, visceral, chemical and sensory nature.

The problem is: the possible influence of consciousness and ideas on the spontaneous motor, visceral, chemical and sensory activities.

During the act of thinking bodily changes of all sorts occur, for while thinking we assume and maintain certain bodily attitudes with simultaneous changes in the spontaneous motor, sensory, vegetative and chemical activities.

The Possible Influence of Ideas.—Under ideas, besides including wishes, cravings, yearnings, etc., I include suggestion, simulation, hypnosis, will power or volition, imagination, and their ilk.

The influence of ideas may be: (a) immediate, occurring at the moment of ideational activity, or (b) remote, occurring later, circuitously, as a final result of ideation. Examples of remote effects of ideas are: insomnia and its dire results, all flowing out of worry; irregular habits of living and the sad effects dependent on personal hygiene and behavior, the result of training and education, which can be reduced to instincts, emotions and ideas. In this way the remote influence may involve all bodily levels—spontaneous motor, visceral, chemical and sensory.

The influence of bodily changes produced may be (a) permanent or (b) transient. Remote influence may sometimes, with other factors, produce permanent bodily changes (such as arthritis or tuberculosis from unhygienic living). But the immediate result is always transient, except in such rare cases as emotionalism leading to the onset of hemiplegia or angina pectoris, etc., in one susceptible to them.

The discussion, therefore, is confined to immediate, transient influence of ideas. This may be (a) direct—without any intermediate link (as when an idea or wish leads to movement of arm or leg); or (b) indirect, with an intermediate link, as when an idea or wish leads to sitting, standing, walking or running, and the latter activity is accompanied, simultaneously but secondarily and accommodatively by visceral and chemical activities.

The possible immediate influences of ideas, wishes, desires, cravings, etc., per se, which are always transient, but may be direct or indirect, are: (a) on the spontaneous motor, locomotor, voluntary or proficient system.

1. Ideas can immediately and directly lead to transient functioning of this system—as in ordinary so-called voluntary activity.

2. Ideas cannot lead immediately and directly to disorders of the locomotor system of a prolonged and continuous nature of the sort described under post-traumatic hysteria. Such functional conditions, not due to gross organic changes, are fatigue or exhaustion states due to microscopic changes, with a psychogenic (ideational and emotional) element superimposed in most cases; or they seem to be continuous but are really transient (coming and going), as the result of inaction with absence of real efforts to produce proper functioning in the involved parts due to blind continuation of an original fatigue state which has since cleared up but in spite of which the patient continues to act by misguided conviction as if the condition of fatigue or exhaustion were still present.

3. In the cases of organic paralysis, whatever power still exists in the partially paralyzed muscles may be developed to its greatest possibility by training, exercise and various physical measures.

(b) On the visceral system.

1. The visceral, vegetative, so-called involuntary or autonomic system can be affected immediately, transiently, but only indirectly as the result of ideas—by primary, initial, preceding, or at any rate simultaneous changes in the spontaneous motor system. Salivary and gastric secretion of so-called “psychic” origin seem to be exceptions to this rule.

2. There is no ideational control, immediately and directly, over the involuntary muscles of the internal organs (heart, etc.). Indirectly, however, through the intervention of ideas, the involuntary system, for example the heart’s action, may be modified, immediately and transiently, by adopting, perhaps unawares, even for a momentary period, certain attitudes or postures, which involve the voluntary locomotor or proficient system first, as in the “emotions” (see below) only to be accompanied instantaneously by certain changes in the visceral vegetative system.

3. Permanent prolonged or continuous changes in the visceral system cannot be reduced by ideas, even indirectly, except in cases in which, as the result of ordinary activity or emotion, an organic condition already existing is lighted up (apoplexy, etc.). This would be a remote, circuitous result and initiated indirectly but not continued by the idea.

(c) On the chemical or metabolic system. Changes of this sort accompany the act of thinking and occur with changes in the spontaneous motor and visceral systems but are not produced, transiently or permanently, directly or indirectly, by ideas in the sense in which these words are used in this discussion. We have a certain control over them, in so far as we control the ideational, spontaneous motor and visceral activities.

(d) On sensory activities.

1. Ideas cannot evoke, in direct or indirect manner, transiently or permanently, sensory phenomena—feelings of warmth, cold, touch, pain, pressure, etc.

2. Only by bringing about peripheral changes as the result of ordinary activity or emotions can any such effects be produced, such as angina pectoris from fear or overwork, with mechanical irritation of the nerve endings and subjective appreciation (consciousness or awareness) of this.

3. By ideas leading to distraction of attention one may inhibit or repress the degree of awareness, consciousness or feeling of sensations of all sorts.

4. But as in the case of continuous or prolonged motor paralyses, so also continuous or prolonged sensory losses cannot be produced by ideas.

Power and Mechanism of the Emotions.—In what we call the emotions, we have reactions at various levels—ideational, locomotor, sensory, visceral and chemical with, at the same time, a varying degree of consciousness, awareness or feeling of certain of these changes taking place. In the emotions are combined both mental and bodily reactions. But hence, too, the views expressed in the foregoing on the limitations of the power of ideas hold true, for the difference between so-called emotions and ordinary voluntary activity, especially when the latter is pronounced and exaggerated, as in running, is in the motive or the mental state present at the moment. Otherwise the *modus operandi* is the same and the reaction in the visceral and chemical systems present in the emotions is produced not directly but indirectly by the mental state, and accompanies or is secondary to activity in the proficient systems which precede it, and which itself is directly produced by the first reaction.

In the emotions there occurs first a change in the mental state—consciousness and ideas, followed in order by spontaneous motor activity, visceral (including ductless glands) and chemical changes.

In the emotions there is no new type of human activity but merely a certain combination of the activities of consciousness, ideation, the spontaneous motor, visceral, sensory and chemical systems, with the special characteristic of a peculiar mental state or attitude which differentiates it from ordinary non-emotional behavior.

Aside from the attainment of a mood of well being and mental calm, obliterating unnecessary, destructive emotions, and permitting a joyful, forward-looking, constructive attitude toward life, the value, possibilities and therapeutic efficacy of all forms of psychotherapy, legitimate or illegitimate, are both physical and mental, for they (a) directly and immediately lessen the drain on consciousness, ideation and the spontaneous motor system; (b) indirectly but simultaneously and immediately, through control of the locomotor system, lessen the drain on the visceral and chemical system; and (c) indirectly and remotely but not immediately overcome the tendency to emotionalism, bad habits and evil results from prolonged continuation of such unhygienic habits of living.

DISCUSSION

PROF. ANTON J. CARLSON: Eight or nine years ago I was quite a regular visitor at the meetings of the Neurological Society and at that time I somehow began to feel that this group was more interested in diagnosis than in etiology. That may have been an entirely erroneous impression. I hope it was.

The subject of Dr. Solomon's paper is a comprehensive one. To do it justice one would have to hold several sessions. It includes, of course, many points that are entirely unsettled, many points which are controversial, on which one may not be dogmatic.

Dr. Solomon said he would discuss the subject primarily from the clinical point of view. This Society is a clinical group and having invited a physiologist to discuss the paper, you will have to take the consequences if he discusses the subject from a physiological point of view. Not that there is any essential difference in the final result. But suppose I draw the picture as I know the subject, physiologically, and you already have the clinical picture, so you can take your choice, or refuse both.

In the first place I think that the title of the paper is not a happy one. I do not accept the mechanistic dualism of the "mind" acting on the "body." And I have for years deprecated that not a few neurologists make a distinction between "nervous" diseases and "mental" diseases. If the "mind" or brain is not a part of the body, what is it? In the physiological vernacular the action of the mind on the body means the nervous coordination of body functions.

As we analyze these diagrams of voluntary movements, movements of the heart, movements of the visceral organs (so far as they are supplied with musculature) and secretions (so far as these are controlled by nerves), we are dealing with nervous processes, leaving out for the moment the factors of chemical control. We are dealing with nervous processes passing out from the brain, the cord and peripheral ganglions and so far as these are dependent on afferent impulses conscious or unconscious, we are also dealing with nervous processes. The interrelation, conscious and subconscious, in the brain of afferent and efferent impulses is likewise a matter of nervous processes; so that from the point of view of a physiologist "mind" or mental action is merely a question of the correlation between the various parts of the body as

mediated by the nervous system. "Mind" in this sense is merely the sum total of conscious nervous processes or ideas going on in the brain at any moment.

I do not like the term "mind" even as defined by Dr. Solomon, because there usually is about it an aura of something mysterious and intangible, something of the stuff that filtered into us from nursery tales and primitive religious instruction. I am afraid that most of us, when we speak of the mind as distinct from the body, picture not only abstract ideas, but add ethereal arms and legs and mouths and noses to that mind as well.

But it seems to me we are on solid ground when we focus on the nervous processes. I do not offer this as the final analysis, but certainly from the point of view of physiology, it is the only avenue of actual progress, as shown both by the past and the present.

Dr. Solomon brought out one question which is quite controversial; namely, the relation of the emotions to ideas—the etiology of the emotions. I am neither a psychologist nor a psychiatrist, and nine-tenths of you probably know more about the subject than I do, but this is the way it looks to me. Some of the emotions may be hereditary. Possibly the emotional phases of appetite, of hunger, of sex, may be essentially inherited. I do not admit that they are. They may be. The capacity to feel or experience emotions is, of course, inherited. But apart from these, the emotional part of life is simply a matter of memory of the kind of relations or experiences we have had in the past with the particular idea or nervous process that happens to be going on in our brain at any one moment.

Let me illustrate my meaning by an example. Assume that we see a snake before us. In people who have been bitten by snakes that may arouse anger or fear. People who have touched snakes and found them cold may shiver. People who have never come in contact with snakes, but who have heard tales of snakes from the nursery up, may have a feeling of nausea: it is an evil animal, associated with the fall of man, and what not. In a primitive child, the snake would draw curiosity or delight according to its color, pattern or mode of motion. To a person who knows snakes, I mean biologically, there is neither loathing, fear, anger nor shivering but possibly a little of the curiosity and delight of those who know nothing else. Hence, the kind or quality of emotions (what we ordinarily call the qualitative phase of ideas) depends on one's individual experience in the past; it is a question of memory, of linking up the past with the present nervous process. The controversial point in the matter is this: whether emotions can be looked on as a memory process alone, or whether it is necessary to have all the bodily components in the original experience present as causes or sequences of the current memory process. Now so far as I know, gentlemen, that question is not yet settled.

You are all familiar with Sherrington's dog. Sherrington selected a very emotional dog; emotional in pleasure and joy, as well as in anger, disgust, etc. This animal was, by transection of the spinal cord and section of as many afferent nerves in the head and neck region as was compatible with life, deprived of most of its proprioceptive and enteroceptive fields, and yet after all that depletion this fraction of a dog retained his original emotions in quantity and quality. That experiment is not conclusive, but it takes more than talk to nullify its weight. To say that it is necessary to have concomitant with the memory processes of emotional tone the action of the cardiac, the secretory, the motor or inhibitory phenomena of the genito-urinary and the gastro-intestinal tracts, etc., that were associated with the cerebral processes initiating the memory complex, is merely labeling an interesting theory as the

proved fact. Do you not agree with me, that the question of the physical reality of so-called imaginary pains is, to say the least, an open one? This is the way the facts appear to me: The physical reality or intensity of any memory process, including so-called imaginary pain, depends on whether these are the sole brain processes for the moment or whether these processes are balanced by the brain processes of critical judgment. In the absence of critical judgment, as in the child, the insane, in hypnosis, or in dream states memory processes or "imaginary" situations pass as real experiences, probably with the body reactions of the actual experiences. Most of us can "concentrate" sufficiently so that "imagination" or the mere memory processes will evoke the thrills of pleasure or the nausea of disgust in experiences remote in years of time and miles of space.

Certain things are pretty well worked out in the subject matter of Dr. Solomon's paper; that is, the nervous relations and reactions that are concerned with our external environment. I mean the skeletal neuromuscular system of normal persons. I do not mean to say that we know as yet all the intricate complications that can come up in this system in functional disorders. The efferent side of the so-called involuntary system is also pretty well worked out, as you all know, thanks to Gaskell, Langley and numerous other workers in this field. We have taken one system after another and determined precisely the kind of motor nerves it has, and how they connect with the brain; the kind of secretory nerves and their paths, etc. Anatomically these things are pretty well worked out. Physiologically the story is less complete. It is a long step in advance to lay bare an efferent nerve and by stimulating it artificially find that it causes secretion or inhibition. But these efferent facts alone do not reveal the complex working conditions of these systems in the intact body either in health or in disease; but it is a great step to know the anatomic details of the efferent mechanism. That is what I mean by this field being fairly well worked out.

Regarding secretory nerves to the glands of the internal secretions there is much literature; but there are few facts. I do not want to be dogmatic, but in my judgment secretory nerves to any gland of internal secretion have not yet been demonstrated. Cushing's work on secretory nerves to the hypophysis fell down. The work of a number of investigators on the secretory nerves to the thyroid gland is in the balance. Ten years ago we thought that the presence of secretory nerves to the suprarenals was proved, but we are less certain of it today. It is an unknown field. The working of the afferent or sensory components of the visceral system is also largely a matter of conjecture. I have a feeling that this system plays important rôles in health and disease in man, and new facts established here may mean more to medical progress than the writing of many books.

I am grateful for this opportunity to listen to Dr. Solomon's paper. We do not agree on some points. That is neither here nor there, provided his theories lead to active investigation.

Now and then there comes a superman who can blaze trails of real progress in a confused field. But we have had too much systematization in this "mind and body" field and too little intelligent work. What I hope is that the author's interest and analytical skill will not end with charts and schemata but in constructive work on patients and animals. Maybe I am prompted a little by selfishness in this connection, as it is clear to me that the rate of completion of our knowledge of physiology of the nervous system depends largely on the scientific interest and industry of the psychiatrist and the neurologist.

DR. SOLOMON: In regard to the use of the term "mind," I did not really wish to use that term. I tried to get away from it by saying that when I use that term, I refer to consciousness and ideas. The reason I presented this subject in a dogmatic fashion was, so to speak, to throw down the gauntlet on various disputed points.

With respect to what Professor Carlson has said about instinct and emotions not being inherited for the most part, I think that what he means was that although the fundamental emotions like fear are really inherited, the attachment of the emotion to any particular thing is acquired in the life of the person and is dependent on experiences.

Experiments by Pavlov and others, rather conclusively prove that secretory nerves to the salivary and the gastric glands exist.

I have endeavored to adopt the clinical point of view in discussing a complex and obscure subject. We need the laboratory worker, we need the physiological worker, and we also need the clinician, who will endeavor to apply in an interpretative and synthetic way, in so far as he is able, the knowledge we have already gained from the laboratory and the clinic. We need to take a stand on these questions.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 26, 1920

SAMUEL D. INGHAM, M.D., *President*

A PATIENT WITH MYOCLONUS FOLLOWING EPIDEMIC ENCEPHALITIS. Presented by DR. S. F. GILPIN.

C. H., aged 45 years, white, an American, with a negative family and past history, about the middle of March, 1920, was ill apparently with a light attack of influenza. He remained at work until March 28, when he had a severe pain across the chest, extending to the testicle and rectum. It continued for three or four days. He soon became delirious; he continued in this state for five weeks, during which time he had muscle spasms and for three weeks he had hiccough. The jerkings continued all over the body and were so severe in the chest and abdomen that he could not rest; this state continued until his admission to Jefferson Hospital, September 14. He had lost about 50 pounds in weight.

Physical Examination on Admission.—The tongue showed a fine tremor. The pupils were equal and reacted normally. The eyegrounds were normal. The deep reflexes were increased; the bilateral Babinski sign and ankle clonus were present. The patient's mind was clear. The Wassermann test of the blood and cerebrospinal fluid and the urine reports were negative.

At present he shows no increase of deep reflexes and the Babinski sign and ankle clonus have disappeared. His chief complaints are the muscle contractions and pain in the left leg and about the testicles and rectum. This pain is paroxysmal, sharp and stabbing and so severe that he cannot sleep. When free from pain he sleeps well, and he has gained considerably in weight. The contractions are general, including those of the muscles of the trunk as well as of the extremities. They occur about seventy-two times a minute; they are not synchronous with the pulse, and their severity has lessened greatly during the past month.

A patient with a similar case was brought into the men's psychopathic ward at the Philadelphia Hospital last winter with a diagnosis of psychosis because of his delirium. He presented moderate temperature and the same marked muscle contractions. The interesting problems in the patient are those of the myospasms, which are rather choreiform in type except in their regularity, and the sharp shooting pains which resemble those of tabes and are distributed like root pains.

A CASE OF BILATERAL EIGHTH-NERVE PALSY; SYPHILIS. Presented by DR. M. A. BURNS.

History.—W. J. H., aged 26 years, a fireman, had a negative history; he denied syphilis. The patient had been married five years and had no children; one induced abortion.

In January, 1920, the patient was exposed to venereal disease, but claimed that he was not infected. Late in February, 1920, he suffered from what his physician called influenza, from which he recovered. During this illness he had a rash which persisted for weeks. The blood Wassermann test was then strongly positive. On Sept. 22, 1920, the patient came to the neurologic clinic at Jefferson Hospital. His chief complaint was deafness of the right ear, of one week's duration, and deafness of the left ear of three weeks' duration. He was dizzy and wobbly when he walked and had occipital headache.

Examination.—There was deafness in both ears and a facial paresis on the right. Romberg's sign was present. The pupils were equal and regular but reacted sluggishly to light and in accommodation. The grip was good. There was no tremor nor atrophy in the upper extremities, and sensation and the reflexes were normal. He was ataxic in both legs. The patellar reflexes were increased; no ankle clonus or Babinski's sign was present.

Laboratory Findings.—On September 22, the blood and spinal fluid Wassermann were +++++. The globulin was markedly increased in the spinal cord, which had in addition 104 cells. Under treatment, which has been vigorous and associated with drainage of the spinal fluid on five occasions, the Wassermann test on November 10 was ++ and the cells in the spinal fluid were 24.

DISCUSSION

DR. W. B. CADWALADER said that Dr. Burns' case was particularly interesting because of the lymphocytosis in the spinal fluid following, or associated with, the attack of influenza. Dr. Cadwalader had seen cases in which there were present the signs of ordinary myelitis, but it was difficult to determine whether they were due to syphilis or influenza; lymphocytosis occurred in both diseases. Dr. Cadwalader thought Dr. Burns' case was syphilitic.

DR. GILPIN stated that the patient gave no history of epidemic encephalitis. In the majority of cases the spinal fluid in this disease did not show lymphocytosis. Considering the symptoms and result of treatment, there can be little doubt as to the diagnosis.

DR. T. H. WEISENBURG said he did not recall seeing bilateral eighth nerve palsies in patients as young as this one, but he had seen nervous involvement in many cases of syphilis within one year after the infection and in a number while the secondary eruption was still on the body. He had seen third and seventh nerve palsies and one bilateral facial palsy within a few months after infection.

Dr. Weisenburg said that he had at the present time a patient with well marked tabes, with the history of an infection at 24, who a year and a half later, while she still had an eruption on her hands, presented pains, disturbance in gait and a beginning optic atrophy. This type of case was unusual. Dr. Weisenburg thought in these patients there was present one of two conditions, either a special predilection on the part of the patient for the specific virus or, what is more probable, a spirochetal infection which had a predilection for the nervous system.

Dr. CHARLES S. POTTS said that he had seen a rash in a case of epidemic encephalitis which covered the entire trunk and resembled very much that of measles. It lasted about a week and then disappeared. Dr. Mills had also seen the patient and had concurred in the diagnosis.

DEMONSTRATION OF A DEVICE FOR SUPPORT IN FACIAL NERVE PARALYSIS. Presented by N. S. YAWGER.

A description with photographs of this device appeared in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, December, 1920, p. 659.

A CASE OF ASTASIA ABASIA. Presented by S. F. GILPIN for Dr. F. X. DERCUM.

L. M., a white man, aged 58 years, complained of vertigo when attempting to walk and of a dragging of the right leg. Seven years ago, while going to work he noticed a paralysis of the right side of the face; he was told that he had had a stroke. When presented he showed evidence of an old Bell's palsy on the right side of the face, with some contracture. Otherwise there was no evidence of organic disease. In attempting to walk he hesitated, shuffled his feet and acted as though glued to the floor. When he was able to start to walk, he walked well, except for some stiffness of the right leg. The paralysis of the right side of the face and a friend's diagnosis of a stroke had apparently been the suggestive factors in bringing about his present nervous condition which was considered functional.

A CASE OF MYOCLONIC LETHARGIC ENCEPHALITIS WITH UNUSUAL PATHOLOGIC MANIFESTATIONS. Presented by Drs. N. W. WINKELMAN and T. H. WEISENBURG.

Clinically, the unusual feature consisted in the pains which were sharply limited to the head, upper limbs, chest and abdomen to the line of the umbilicus and never below. The pathologic findings were those which are usually present in this disease, but careful study of the spinal cord demonstrated that there was a lessening in the intensity of the pathologic process as the lower levels of the cord were reached, and that the spinal cord below the lower thoracic level was altogether normal.

The patient was a physician, aged 40, with a history of influenza in February, 1920, seen by Dr. Weisenburg, April 22, 1920. Shortly after having had the influenza the patient complained of dizziness, which was not very severe. About a month before he was seen, he had hiccough which lasted for three days. A week later he complained of headache, the pain being all over his head and in his ear. Two weeks before he was observed, he began to complain of twitching in various parts of the body, first in the arms, then in the legs and

finally all over the body. Soon after, pains appeared and were described as sharp and shooting, but were never present below the line of the umbilicus. Two days before death, he became delirious and had visual hallucinations. When examined by Dr. Weisenburg on the day of his death, with the exception of visual hallucinations, he was normal mentally and discussed his case with some knowledge of the disease, although he insisted that he would get well. The twitchings were present chiefly in the shoulders, chest and abdomen, but were not at all marked in the lower limbs. There was considerable diaphragmatic twitching. The twitchings were of the character seen in Huntington's chorea, but were much more rapid. The limbs were not weak but the deep reflexes were increased. There was no Babinski sign, and there were no ocular palsies. He was seen at 5:15 p. m.; the twitchings rapidly increased, and he died suddenly the next morning at 4.

DISCUSSION

DR. WILLIAMS B. CADWALADER said he had recently had a case of influenzal myelitis. The patient was paralyzed both for sensation and motion below the level of the costal border, but after some weeks there was almost complete recovery. Hyperesthesia at first was intense throughout the trunk and lower limbs; it seemed to be analogous to the hyperesthesia and pain sometimes seen in poliomyelitis, for it was relieved by lumbar puncture. The exact cause of the pain was not clear, but it is reasonable to suppose that there had been much swelling and edema of the cord, like that found by Dr. Winkelman. On this account the posterior roots might have been pressed against the vertebral wall becoming irritated, and thus caused pain.

Gordon Holmes has reported cases of gunshot wound in which he found a great deal of pain or hyperesthesia that he attributed to intense swelling and edema of the cord caused by contusion from the impact of a bullet, although in some of his patients the roots also were injured. Dr. Cadwalader suggested that the same explanation for central pain, as described by Holmes, might be given for the pain and hyperesthesia seen in the early and acute stages of influenzal myelitis and poliomyelitis. After withdrawal of the spinal fluid, Dr. Cadwalader thought the pain subsides in a striking manner. The pain in the muscles associated with repeated involuntary contractions seen in the late stages of epidemic encephalitis was of a different character and separate origin.

DR. S. P. GILPIN said that in his case the first symptom was pain of such intensity that it drove the patient to the hospital. The delirium developed within four days after onset of the pain, and the muscle spasms developed while the patient was delirious. He thought Dr. Cadwalader's explanation of the origin of the pain was good, but in his patient the pain was still very marked, although several months had passed, and the patient had almost recovered from the myospasm.

DR. WEISENBURG said that as to the assumption of a resemblance between the pains of poliomyelitis and encephalitis, he was of the opinion that there was not the slightest ground for this. He had made what is probably the only neurologic study of a poliomyelitis epidemic, in 1916, when he reported 717 cases. As a result he concluded that in every acute case of poliomyelitis there was present a limited amount of meningitis, and that the pains in the limbs were the result of this. In fact, one could distinguish the paralyzed limbs because they were painful on pressure. Such a condition does not exist in encephalitis. Besides, the pains described were of a different character. In

encephalitis they are sharp and shooting; in poliomyelitis, while there are occasional sharp pains, the patients chiefly complain of pain in the head and neck and on pressure over the limbs.

AN INTERESTING CASE OF MYELITIS. Presented by DR. F. B. MARSH.

This particular case was one of the many conditions in which influenza evidently played an important part.

A white man, aged 55 years, an engineer, was admitted to the Pennsylvania Hospital, Sept. 27, 1920. He complained of wasting and loss of strength in the muscles of the arms and shoulders and jerking of the muscles of the same parts. He denied syphilis. About five years ago the patient had had a stroke of apoplexy. He fell over, not unconscious, but his right arm and leg were paralyzed and he was unable to speak. These symptoms lasted only twenty-four hours, but his arm and leg were weak for about two years. His condition was good until about two years ago when he developed influenza. On recovering from that disease he realized that his shoulder muscles and the muscles of his arms were greatly atrophied and were practically powerless. There were no other symptoms. His grip was good. About one year ago he noticed that various muscles of the shoulder and arm jerked at times, and that his gait was slightly unsteady. His general condition had been good.

Examination.—The face movements were normal. The pupils were equal and very irregular; the right reacted to light sluggishly, the left more promptly. The tongue protruded in the midline. There was no paralysis of the vocal cords and speech was normal. The neck showed muscular atrophy and loss of power in the posterior and lateral groups of muscles. The trapezius and sternomastoid muscles were fairly well preserved. The patient was not able to lift his head up slowly, but had to throw the head back and hold it there by shifting the center of gravity. There was complete atrophy of all of the muscles of the shoulder girdle including the biceps. The forearms and hands were fairly well developed with good power in them. Biceps reflexes were absent; the triceps reflexes were exaggerated, especially on the right. Twitching of the muscles of the shoulder girdle was noted at times. The lower extremities were well developed and slightly spastic. The knee jerks were much exaggerated, but equally so. An ankle clonus was present on the right. There was no constant Babinski sign. There was no disturbance to heat or cold, but pain sense was apparently diminished in the right axilla. Electrical reactions were absent in the atrophied muscles. The triceps reacted normally. The roentgen-ray report on the cervical spine was negative. The urine, blood and spinal fluid were normal except that the Wassermann test on the blood was weakly positive on two occasions.

Dr. Marsh thought the most probable cause was either influenza or influenza with syphilis. An influenzal myelitis could have developed during the influenzal attack which just antedated the onset of the symptoms. On the other hand, the influenzal attack might have lighted up a preexisting syphilis. The fact that the patient's blood on two occasions gave weakly positive reactions would favor the latter view, while the negative spinal fluid Wassermann reaction would be against it. The nerves supplying the paralyzed muscles arise from the second to the fifth cervical segments. The preservation of the sternomastoid and trapezius is explained by the double nerve supply—the cervical nerves and the spinal accessory. The spasticity of the lower extremities indicates that the myelitis was not complete but involved, either by pressure or infiltration, the lateral columns.

DISCUSSION

DR. MILTON K. MEYERS thought that often a previous injury or disease might obscure the diagnosis in such a case. Among returned soldiers he had found four cases of amyotrophic lateral sclerosis or of progressive muscular atrophy, which in some of the cases were unsuspected by the claimants; in some the nervous disease was attributed to injury. In the present case there was a spastic condition of the lower extremities which might well be explained on the basis of the patient's previous hemiplegia. It seemed to Dr. Meyers that the condition of the upper extremity could be explained as a progressive muscular atrophy. Absence of sensory symptoms would indicate this.

DR. N. W. WINKELMAN said this was a case in which localization was obtained not from sensory, but from motor symptoms. There was absence of biceps reflexes, with biceps atrophy, representing the fifth cervical segment, with preservation of the triceps reflexes, the sixth cervical segment, so that the lesion was above the sixth cervical segments. Since the muscles of the back of the neck were distinctly atrophied there must be involvement of the second, third and fourth cervical segments. The patient's answers as regards objective sensation were contradictory but there were sensory disturbances, though these were not as marked as the motor disturbances. The lesion extended from the second to the fifth cervical segments, even though the diaphragm was not involved, since the lesion was probably incomplete.

Book Reviews

THE PSYCHOLOGY OF FUNCTIONAL NEUROSES. By H. L. HOLLINGWORTH, Associate Professor of Psychology in Columbia University. Cloth. Price, \$2 net. Pp. 259. New York: D. Appleton and Company, 1920.

This is an interesting and able work, based on the observations by the author of approximately 1,000 cases in U. S. Army General Hospital No. 30 at Plattsburg Barracks. After consideration of many theories, he concludes that the mechanism of redintegrative response affords an explanation for the reactions of the psychoneurotic patient. He describes redintegration as "that type of process in which a part of a complex stimulus provokes the complete reaction that was previously made to the complex stimulus as a whole." He then discusses normal and abnormal redintegration, and points out that there are three levels of response discriminated in the reaction from a given stimulus or situation: (1) the postural or cerebellospinal level, (2) the cortical level, and (3) the autonomic level. In the psychoneurotic patient, because of more or less mental inadequacy or stupidity and lack of sagacity, there is not a normal redintegration in the cortical, autonomic and postural levels, especially in the spinal and autonomic levels.

In the mental ratings of psychoneurotic patients, the author's data consist of intelligence tests of nearly 1,200 patients. The mental age ranged from 8.3 years, in cases of mental deficiency, to 13.2 years in psychasthenia. The epileptic and hysteric groups had a mental age of about 12 years. The mental age of the normal soldier is not over 14 years.

The author states that "it is clear that the difference in specific symptoms is correlated with a difference in mental level," and that the lower the intelligence level, the more liable the soldier is to display the so-called conversion type of hysteria, and the higher the scale of his intelligence, the more likely he is to become neurasthenic or psychasthenic. Officers, for example, rarely show hysterical symptoms, usually having the so-called anxiety neuroses.

The author concludes that it is this predisposition toward redintegrative conduct that constitutes or characterizes what may conveniently be called the psychoneurotic constitution.

Finally he summarizes his conception of the functions of psychologic service in a neuropsychiatric hospital: "Through the intelligence examination to throw light on the clinical condition, the complete diagnosis, the proper disciplinary measures, the military or civil serviceability, and the most effective and expeditious disposition of patients. Through the further analysis of such data to make at least a suggestive contribution to the study of the factors operative in the production of the psychoneuroses. Through the inventory of the patient's special aptitudes and educational equipment, to place him effectively for maximal therapeutic occupation. In a similar way to afford him aid in vocational adjustment and in the development of purposiveness and aim, in the effort to make of him a more balanced and a better adjusted personality. Through graphic records of the increments of functional capacity under physio- and mechano-therapy, to portray for his own encouragement and for the information of the physician the course of progress. Through inten-

sive individual reeducation to direct and stimulate the patient in his recovery from specific symptoms and disabilities, thereby improving his general morale and his attitude toward the hospital and the service. Finally, through special experimental technic, to demonstrate the degree of rehabilitation and the approach to complete recovery in particular cases, in exact and comparable terms."

When the war broke out psychologic examinations were instituted in various camps. Later the psychologic corps, which at first was part of the neuropsychiatric division, was made an independent unit, for the first time in the history of any army. In connection with this notice of Dr. Hollingworth's book, it may not be amiss to give the point of view of most of the neuropsychiatrists who had actual experience with the psychologic examinations, working more or less with the author and his associates.

In analyzing the data and conclusions of Dr. Hollingworth it is interesting to note that he had, in his 1,172 cases, 339 epileptic patients, about one third of the total number. He, of course, was not responsible for the diagnoses that were furnished him, but it developed that most of these so-called epileptic patients really had hysteria. In the author's studies the mental age of epileptic and hysteric patients was similar. This is pointed out only to show that some of Dr. Hollingworth's conclusions were based on incorrect data.

Medical officers in Plattsburg regarded the psychologic service as of considerable value in the aid that it gave them in the mental examination of patients, saving them a great amount of work. The chief value of the psychologic service, however, was that it had workshops and other methods of keeping patients busy. But it is worthy of note that comparatively few soldiers would go to the shops voluntarily; most of them had to be ordered there. After the armistice the psychologic service was practically of no value, for the soldier had only one object in view and that was to be discharged. All this is said not to disparage Dr. Hollingworth's work, but to give the reader the point of view of the soldier and medical officer.

The value of the psychologic service in the army varied in different camps. For example, in one camp, in which the psychologists recommended for discharge all those whose mental age was 9 or 10 years, careful inquiry by the neuropsychiatrists showed that a large percentage of these men, although they had low mental rating, were able to get along in their civilian occupations and to support themselves and their families. In other words, they were relatively adaptable and efficient. On the other hand, there were cases accepted by the psychologists with a mental rating of from 10 to 12 years, who were found to be definitely handicapped, so far as their ability to earn a livelihood was concerned; they gave a history of shifting from one occupation to another and being unable to support themselves; they manifested, to a large extent, many reactions common to constitutional psychopathic persons.

Psychologic work went to the discard, however, after the March, 1918, offensive of the Germans, for then it was a question of sending many men abroad as rapidly as possible. The further the war progressed, the less the psychologic corps gained in favor and by the army as a whole it was regarded as an encumbrance. It is questionable whether in a future war, a psychologic department would be found in the Surgeon-General's Office. Every soldier of any experience knows that either a corporal or a sergeant of a company knows more of the individual merit of a soldier and what particular thing he can do best, than would be indicated by the most expert psychologic examination.

No doubt a psychologic examination can give the mental level of a man, but it cannot indicate the fighting level, for mental age does not with any accuracy indicate the vital factors of temperament, moral attitude, industry, obedience and other qualities in the make-up of a good or poor soldier (or civilian).

FUNCTIONAL NERVE DISEASE. An Epitome of War Experience for the Practitioner. Edited by H. Crichton Miller, M.A., M.D., Formerly Medical Officer in charge of Functional Cases, No. 21, General Hospital, Alexandria; Late Consulting Neurologist, 4th London Hospital. London: Henry Frowde, Oxford University Press; Hodder & Stoughton, Ltd., Warwick Square, E. C. 1920. Price, \$4.50. Pp. 208.

The chief value of this work is for the general practitioner for whom it was written. For the neurologist it is interesting and instructive as presenting today's British thought on the psychoneuroses. As the book consists of twelve chapters by eleven authors, each writing from his own point of view, there is a good deal of disharmony and the composite gives the impression of heterogeneity. Indeed, an oldish observer has a feeling of being present at the birth of English neurology—in so far as it pertains to the psychoneuroses. The various chapters (well written, clear and helpful) represent fairly well sundry stages and phases in the development of neurology heretofore passed through by other countries. The great war at once fairly forced functional nervous disorders on medical England, and with the bull dog's customary slowness, tenacity and ultimate effectiveness medical England tackled the job. Consequently, this little book contains a good presentation of the psychoneuroses of war in their various relations and as looked at from different sides. Every chapter is good, though here and there the work is unmistakably immature. The chapters on "Psychoanalysis," "Repression and Suppression," "Regression and the Mother Complex" will be interesting and illuminating for many who have not already delved into these things, and assuredly will give them new and useful ideas on functional nervous disorders. Chapter 12, by William McDougall, is masterly. His task was to summarize the preceding eleven chapter. This he does not do; nobody could. But in seventeen pages he gives a remarkably clear and reasonable exposition of the psychoneuroses, the principles which should govern their treatment and the mechanism of their recovery.

The assumption by the editor that what we have learned of war neuroses will be useful in the estimation and treatment of peace neuroses assuredly is well founded.

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A STUDY OF SOME PECULIAR CHANGES FOUND IN THE OXONS AND DENDRITES OF THE PURKINJE CELLS *

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INTRODUCTION

A peculiar balloon-like swelling of the dendrites of the ganglion cells was noted first by Schaffer¹ in amaurotic family idiocy. Later the same condition was observed in the apical dendrites of the Purkinje cells by Rogalski, Jansky, Schob, Bray, Sachs and Strauss,² Schaffer and others. This swelling of the dendrites was considered by these authors as one of the pathognomic findings of amaurotic family idiocy.

In 1906, Sträussler³ described the same kind of alteration among the dendrites of the Purkinje cells, together with similar swellings of the axis cylinders in a psychosis—in a woman 36 years of age—which manifested certain cerebellar symptoms associated with mental agitation and intellectual weakness. The cerebellum of this patient showed congenital malformation—defect of the granular layer. He attributed this peculiar change of the dendrites to their incomplete development and overwork.

In 1910, Sträussler, in a report on three cases of juvenile general paralysis, described a hypoplasia of the cerebellum and peculiar swellings of the dendrites and axis cylinders. He thought that these swellings were identical with those of amaurotic family idiocy and also with those shown in his previously described case. He believed, as a result of his studies, that there is an intimate relationship between juvenile general paralysis and hereditary family diseases, based on developmental defect of the central nervous system.

* From the Pathological Laboratory of Danvers State Hospital.

1. Schaffer: Zum normalen und pathologischen Fibrillenbau der Kleinhirnrinde, *Ztschr. f. d. ges. Neurol. u. Pathol.* **21**: 1, 1914.

2. Sachs and Strauss: The Cell Changes in Amaurotic Family Idiocy. *J. Exper. Med.* **12**: 685, 1910.

3. Sträussler: Ueber eigenartige Veränderungen der Ganglionzellen und ihrer Fortsätze im Centralnervensystem eines Falles von kongenitaler Kleinhirnatrophie, *Neurol. Centralbl.* **25**: 194, 1906.

So far as the writer has been able to determine from the literature these peculiar swellings of the dendrites have never been found in any other diseases than those mentioned above. As etiologic factors, most of the authors seem to favor congenital weakness, endogenous factors such as suggested by Sachs and Strauss in amaurotic family idiocy, and some external agent, such as overwork.

In the experimental study of Ramon y Cajal⁴ the injured dendrites of cerebral as well as cerebellar cells showed, as a rule, no regenerative reactions; but in one young animal experimented on, the dendrites of the Purkinje cells presented various nodular enlargements. Their reaction to the injury was also shown by a change in diameter, as well as in the form, length and structure of the secondary and tertiary branches.

Swellings of the axis cylinders have also been noted by various observers, and the explanations of the nature of these have been almost equally varied.

Cajal was the first to describe the swelling of the axons of the Purkinje cells resulting from experimental lesion. He called attention to certain terminal sacs (*les boutons terminaux*), some belonging to Purkinje cells, others to afferent fibers of the white substance. Most of these sacs were situated rather far from the lesion in apparently healthy tissue, while in the neighborhood of the lesion Purkinje cells were markedly degenerated, presenting a granular appearance, with no trace of intracellular neurofibrils. This led Cajal to believe that the sac formation is a reaction of the living protoplasm, and that it represents a regenerative process of the axons, whose continuity has been disturbed.

Rossi also observed the same interesting changes among axons of Purkinje cells in a case of cerebellar sclerosis. The axon was replaced by a round or oval mass, single, more rarely double, of homogeneous appearance, though sometimes showing within it a few fibers. This mass was generally encountered within the granular layer at a distance from the Purkinje cell, to which it was attached by a filament. He found also similar masses at the level of Purkinje cells where the latter seemed to be lacking. Most of the Purkinje cells exhibiting these changes were smaller and presented fewer dendritic arborizations. Some of these swellings were provided with processes which often threaded their way to the molecular layer in which they mingled with other nerve ramifications, thus rendering it impossible to determine the manner in which they terminated. Rossi interpreted the latter condition as a regenerative process of the preexisting normal collaterals.

4. Cajal: *Histologie du système nerveux de l'homme et des vertébrés*, traduite de l'Espagnol par le Dr. Azouley, 1909.

When the integrity of the Purkinje cells has been impaired by some cause or other, they no longer possess the ability to regenerate completely, and therefore they try to reproduce some other paths for their compensatory efforts. According to Rossi, this is the most important phenomenon from the point of view of the function of the organ.

Later Marinesco⁵ described structures identical with those described by Cajal and Rossi. He reported cerebellar symptoms had been shown clinically in one case, and at necropsy a large cystic cavity was found in the cerebellum. In another case a cerebral tumor, involving the left auditory nerve and severely compressing the cerebellum, was found at necropsy. One patient, a man aged 67, exhibited areas of softening in the cerebellar cortex, while the remaining patient had tubercle deep in the white substance of the cerebellum. Clinically, this last patient was a victim of Pott's disease. Marinesco summarizes his conclusions on the study of these cases as follows:

The fibers of the cerebellum, like those of the medulla, show a decided tendency to grow, if interrupted. We have seen them penetrate the interior of the softened region to great distances. Those which have not been able to penetrate have given forth new fibrillation at the limits of the healthy tissue. This zone can, then, from all points of view, be compared with the central end of a divided peripheral nerve. As to the other phenomenon which we have discussed as "*boule des axones de Purkinje*," the swelling of the fibers, the hypertrophy of the neurofibrillar plexus, whether dendritic or axonal, etc., these may be classed as phenomenon of nerve regeneration. Some authors class them among the regenerative phenomena, others regard them as of a degenerative nature. In our opinion they do not represent anything but a special reaction of the nerve cell and fibers, due to a disturbance of nutrition.

In 1914, Schaffer studied the normal and pathologic neurofibrillar structure in material from normal cerebella, amaurotic idiocy, tabes, taboparalysis and senile dementia. He described a peculiar swelling of the axons in the Purkinje cells in all pathologic groups; in amaurotic idiocy there was marked swelling of the dendrites. This differed from the earlier descriptions of amaurotic family idiocy by Sachs and Strauss. These observers did not note axonal changes in the Purkinje cells, though they did state that "the apical dendrite of pyramidal cells and axons were rarely affected," adding that they had "no theory as yet to account for this peculiar selective activity of the degenerative process." Schaffer observed two types of local swelling of the axons: One is stained pale, showing a loosened neurofibrillar structure; the other is dark and homogeneous, characterized by an argentophilic condition. In the cerebellum, therefore, the neuron is affected in all its constituents, that is, not only the cell body with dendritic arborization,

5. Marinesco: Nouvelles contributions a l'étude de la régénérescence des fibres du system nerveux central, J. f. Psychiat. u. Neurol. **17**: 131, 1913.

but the axon. In addition to focalized enlargements of axons, Schaffer described diffuse hypertrophies and atrophies of the axon of Purkinje cells. Schaffer interprets the focalized swelling of the axon as resulting from two causes: first, from hypertrophy of the axoplasm, and second, from the loosening of the neurofibrils following a solution of the interneurofibrillary substance (Axonkittsubstanz). This process is followed by a local deposition of the waste products of pathologic metabolism, which is suggested by the general argentophilic condition of these portions. These peculiar changes of the axon were associated with degeneration and diminution of the tangential and basket fibers in cases of tabes and taboparalysis.

In a case of congenital atrophy of the cerebellum, Sträussler described the swelling of the axons as well as of the dendrites. He attributed both of these conditions to the wasting of the cell and to certain congenital predisposition. Sträussler also observed the same peculiar swelling of the axon in juvenile general paralysis.

In 1918, Professor Kure, Dr. Hayashi of the Tokio Imperial University, and I, observed the same peculiar swelling of the axons in three cases of senile dementia, and reported them at a meeting of the Tokio Medical Association. At the time we suggested that they were probably common in the cerebella of senile dementia.

The swelling of the dendrites and axis cylinders have thus been reported by various observers under various pathologic conditions, and their opinions concerning the nature of these swellings vary. This study was made to endeavor to determine in what diseases these changes are likely to be found, and what significance they may have.

METHOD OF EXAMINATION

The material for this study was secured from brains of the Danvers State Hospital Laboratory series. These brains had been preserved in 14 per cent. formaldehyd solution. Particular care has been taken in the selection of suitable material, especially with respect to proper fixation and the time intervening between death and necropsy. Brains not hardened well and those that came to necropsy more than twenty-four hours after death have been excluded from this study. Small pieces have been taken from the upper and lower vermes, superior and inferior lobes of both hemispheres. Frozen sections have been made at 5 microns and stained by the Bielschowsky method. Ten slides have been studied from each block. Each preparation has been observed under low and high magnifications and the entire section studied. The sections contained, on the average, ten cerebellar foliae. Thus, 600 folia from each cerebellum have been carefully examined for the particular neurofibrillar changes.

In addition to Bielschowsky's method sudan III, thionin, Scharlach R., Weigert's glia staining, and other general staining methods have been employed.

HISTOLOGY OF THE CEREBELLUM

In the study of this problem consideration of the normal histology of the cerebellum cannot be omitted, more particularly the knowledge of the nerve fibers which are closely related to the Purkinje cells and their prolongation. I shall therefore first summarize the normal histology that has hitherto been described by various authors, particularly by Cajal; second, I shall give my own observations, which are more or less different from those already described.

It is convenient to describe the finer structure of the cerebellum under the following heads: the molecular layer, the intermediate or Purkinje cell layer, the granular layer, the white substance, and the basket and cushion fibers.

The molecular layer is occupied by small and large nerve cells. The large cells are found in the deeper part of this layer. The axis cylinders of these large cells have a certain relationship to the Purkinje cells. They run horizontally over the Purkinje cell layer, giving off collaterals at regular intervals. They finally approach one of the Purkinje cells, arborize around it, and form a kind of basket (Koelliker and Cajal). The collaterals also form baskets around the Purkinje cells. These cells of the molecular layer are called, accordingly, basket cells.

At the lower limits of the molecular layer, that is, in the intermediate layer, are found a large number of Purkinje cells, the largest in size of the elements of the cortex, and from a physiologic point of view considered as playing the most active part in the functions of the cerebellum.

The Purkinje cells possess dendrites that run through the whole thickness of the molecular layer. This cell is a voluminous spherical or ovoid body. It has a fibrillary structure. The fibers seem to wind around the nucleus, then turn away to course to the peripheral prolongations. It is worthy of note that the Purkinje cell contains, in comparison with other large cells of the brain, spinal cord, optic thalamus, etc., and very few pigment corpuscles. The rod shaped Nissl bodies are arranged in circular order around the nucleus, those in the base being larger than those in the apex. The apical dendrites also show rod shaped Nissl bodies. In silver preparations one can observe the collaterals of the axis cylinders which have a tendency to turn back toward the surface of the cerebellum, arborize around the neighboring Purkinje cells and continue further into the molecular layer. The axis cylinders go down into the white matter without diminution of caliber.

It is generally accepted that all the axis cylinders of the Purkinje cells pass through the cerebellar cortex to terminate in the cerebellar ganglions of the white substance. From the apex of the cell a short thick main stem of the dendrite is usually given off, which soon divides into two main stems that extend horizontally in opposite directions. From these two branches many other smaller branches are given off which extend toward the surface of the cortex, these in turn giving rise to many still finer branches which penetrate through the entire molecular layer. The atypical form of Purkinje cell, described by Cajal and others is triangular, conical or often star shaped, and is usually located in the molecular layer. Schaffer described this atypical form of the Purkinje cell in which he occasionally found two nuclei, one smaller than the other. The nucleus of this atypical form is, according to Schaffer, often of oval shape, while the nucleolus is always spherical.

Schaffer described more accurately the relation of the axis cylinders to the cell body. The axons, it is asserted, do not always spring from the base of the cell body, but sometimes originate in the lateral periphery. They arise from a conically shaped elevation of the protoplasm, immediately become very thin and stain faintly. A short distance from the cell the fiber suddenly becomes thicker and stains more darkly. This increased caliber of the fiber is probably due to the beginning of the myelin sheaths. Schaffer observed the collaterals of the axon mostly at a great distance from the Purkinje cell body, given off at acute angles and in directions contrary to the general course of the axon, noting also a bridge of some plasmic substance between the axonal stem and the collateral branch.

The granular layer is almost entirely composed of an agglomeration of small cellular elements of spheroidal shape. Each one possesses protoplasmic prolongations and an axis cylinder. The protoplasmic prolongations are three or four in number; they are short and thin, and, with few terminal branchings, they end in the granular layer. The axis cylinders of these cells ascend in the molecular layer and divide in a T-shaped manner, the branches extending horizontally and terminating freely among the end-arborizations of the dendrites of the Purkinje cells. In the granular layer, there are also several kinds of large nerve cells. Schaffer differentiates two kinds: one, a spindle form in the Purkinje cell layer, the other, a star shaped or multipolar cell, located in the deeper layer. The spindle shaped cells are of two types: one located in a horizontal position in the Purkinje cell layer with its long axis horizontal, and the other located in the upper part of the granular layer and disposed in either an oblique or in a perpendicular position. The dendrites of the first type share in the forma-

tion of the so-called cushion fibers. The prolongations from the lower pole of the second group arborize in the granular layer, while the prolongations from the upper pole mount into the molecular layer, mixing with the basket fibers of the Purkinje cells. The star shaped cells are rather large and possess a great number of prolongations which arborize freely in the immediate vicinity of their own cell bodies, thus forming a sort of network in the meshes of which a large number of granular cells may be enclosed. Some of these prolongations, however, mount upward to terminate in the molecular layer. Schaffer also described a peculiar basket formation around those nerve cells located near the Purkinje cells—a formation similar to that found around the Purkinje cells.

The white substance is formed by a mass of myelinated fibers which extend in opposite directions; one set of fibers is centrifugal, the other centripetal. The centripetal fibers are of two kinds: the mossy fibers of Cajal and the climbing fibers of Cajal and Koelliker. The mossy fibers arborize in the granular layer and enter into relation with granular cells. The climbing fibers ramify principally in the molecular layer and terminate at the dendrites of the Purkinje cells. The centrifugal fibers are entirely derived from Purkinje cells. Schaffer, Bielschowsky and Wolff observed and described the climbing fibers. Schaffer, however, asserted that the ascending fibers did not always follow the course of the dendrites of Purkinje cells but sometimes left them to terminate in the gray substance of the molecular layer. After leaving the dendrites, they take either a horizontal course or an oblique or perpendicular course, attaching themselves transitorily to the dendrites and again losing themselves in the molecular layer.

Basket and Cushion Fibers.—The Purkinje cell layer is a place of rendezvous of fibers of different origin. These fibers arrange themselves chiefly in a vertical direction, that is, they extend from the base of the cell toward the main dendrite. Many fibers run partly in an horizontal, partly in an oblique, direction. These fibers thus form a kind of envelope around the Purkinje cell, as well as a cushion-like support. The enveloping fibers are called basket fibers and the fibers that extend horizontally are called cushion fibers. The basket fibers are derived, according to Cajal and Koelliker, from the basket cells of the molecular layer. Schaffer described these fibers accurately and illustrated them in his paper. The following are the important points of his description: Each collateral of the axis cylinders of the basket cells traverses the main dendrite and cell body and after sending a branch to the right and one to the left, sinks deep into the cushion fiber layer (*Polsterfaserschicht*) and becomes a part of the constituent elements of the cushion. It then runs either to a neighboring Purkinje

cell to take part in the formation of its basket, thus uniting two neighboring Purkinje cells, taking its course in the cushion fiber layer, passes on for a distance corresponding to two or three Purkinje cells, and there terminates around a Purkinje cell; meanwhile its caliber gradually diminishes. Schaffer also states that some of these collaterals, after they have shared in the formation of the basket, pass into the granular layer, the fiber accompanying an axon of the Purkinje cells, then take a sharp turn again finding their way to the fiber basket of the same Purkinje cell, while the others assume a curved course to join the fiber basket of remote Purkinje cells. Ascending fibers were also described by this observer: fibers from the granular layer to the elements of the basket, such as fibers from the spinocerebellar tract, dendrites of Cajal's star shaped ganglion cells of the granular layer and screwlike, winding, thick fibers, their origin unknown but probably derived from the white matter. Some observers have described direct communication between the fiber basket and the Purkinje cell body, but Schaffer contradicts these authors.

PERSONAL OBSERVATION ON NORMAL HISTOLOGY OF CEREBELLUM

Site of Purkinje Cells.—The Purkinje cells are usually found in the intermediate zone, that is, between the molecular and granular layers. A small percentage of these cells are found in the molecular layer, usually immediately above, and occasionally fairly far from, the normal location of the Purkinje cells (Fig. 1). Since this condition of dislocation of the Purkinje cells is commonly found in normal cerebella, it cannot be regarded as an heterotopia. If, however, Purkinje cells are found in the deeper parts of the granular layer or in the upper parts of the molecular layer, an interpretation of heterotopia is justifiable.

Atypical Form of Purkinje Cells.—Although the normal form of the Purkinje cells is spherical and somewhat depressed like a lens or the seed of a pumpkin, it presents several other forms. In the early part of the development, as shown in my study of several fetal brains, there is often a resemblance to a boat, the long axis being horizontal and situated partly in the molecular and partly in the granular layer. Some cells, however, are fairly far above the normal location in the molecular layer and wholly within this layer. The two extremities of the boat are dendritic prolongations which approach nearer and nearer in the course of development, finally meeting and forming the apical dendrite. There are, therefore, in the normal fully developed cerebellum, many transitory forms between the original and fully developed types. Some Purkinje cells have two main dendrites given off from the side of the cell body, while others present two main pro-

jections at the apex of the cell whose courses are in opposite directions, giving the cell the appearance of a uterus with tubes. Those cells which possess a single main stem also show a varied manner of branching, some dividing directly above the cell body, others at a distance from the apex, some having branches of equal thickness and others having branches of considerably different caliber. Purkinje cells found out of their normal position display more or less atypical forms in the general shape of the cell body, as well as in its mode of branching. A small number of cells, however, although normally located, present atypical forms even in normal cerebella. The small dendrite sometimes seen at the side of the cell body is regarded by some authors

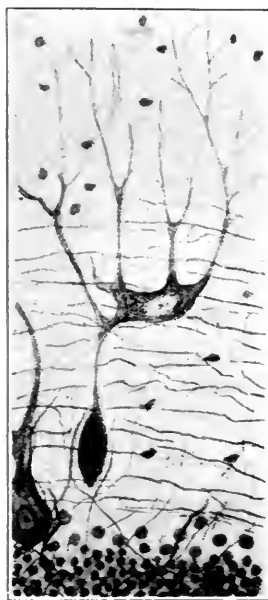


Fig. 1.—An abnormal location of the Purkinje cell whose axis cylinder is swollen.

as a pseudodendrite. The writer does not consider this a good term, since this is only a transitory stage in the formation of the apical dendrite and is purely a developmental characteristic. This, it seems to me, is important for the interpretation of the nature of the peculiar changes found in the cerebellar cortex.

The original embryonic shape of the Purkinje cells may assume in the course of development a variety of shapes—conical, stellar and bizarre forms. The radiating dendrites from the several parts of the cell body may at times be as many as four or five in number.

Dendrites of the Purkinje Cells.—The manner of arborization for the main stems has already been described. The Purkinje cells located in the sulci between the folia show generally an irregular arborization. Some Purkinje cells have dendrites that extend to great distances, while others possess short branches that terminate comparatively close to the cells. The dendrites given off from the sides of the cell body, which some authors regard as pseudodendrites, are sometimes very thin and have a few secondary branches which do not appear like ordinary branches of dendrites. The protoplasmic bridge between the two dividing branches, described by Schaffer, is frequently found, and should not be considered as pathologic. The spindle-like thickening of the main dendrites, which gives rise to a number of small arborizations, is also not a rare finding in the normal cerebellum. But when these swellings are found together with obvious pathologic changes of the bodies of the Purkinje cells or their dendrites, it is sometimes difficult to decide whether they are pathologic or normal.

The Axis Cylinders of the Purkinje Cells.—A knowledge of the normal anatomy of the axis cylinder is especially important for the study of our problem. The axis cylinders of the Purkinje cells are usually difficult to demonstrate, although in pathologic conditions they are likely to be increased in thickness, which makes their course and termination easier to follow. For this study of the normal structure, however, pathologic changes in axis cylinders or tissues from manifestly pathologic cerebella cannot be regarded as suitable. For the study of normal axis cylinders cerebella were used from three patients with manic depressive disease, from three with dementia praecox and from two subjects that were not insane, all in the third to fourth decades. While most of these cerebella were derived from subjects dying of psychoses, there were no gross pathologic changes in either cerebellum or cerebrum and no outstanding pathologic alterations in the cerebellum of any of the subjects.

The proximal portion of the axis cylinder is very delicate and consists of axoplasm and neurofibrils. After a short distance it increases in thickness, due to an addition of the so-called "Kittsubstanz," (gymnaxostroma and myeloaxostroma of Bielschowsky and Wolff). I have found that not all axons of the Purkinje cells enter the white matter. A certain number of axis cylinders, after extending a short distance into the granular layer, turn back toward the surface of the cerebellum in a bowlike curve to arborize around its immediate neighbor or perhaps around more distant Purkinje cells. When the axis cylinder arborizes around neighboring Purkinje cells, it soon gives off a number of collaterals which seem to take part in the formation of the baskets. Some of these axis cylinders, even after reaching a fairly deep part

of the granular layer, turn back to terminate around the neighboring Purkinje cells. It may be questioned whether or not we are here dealing with collaterals of the axis cylinders instead of with the main stems. The caliber of the fibers, however, is always the same, and they show no abrupt turn in their course. Moreover, we do not normally see collaterals in the immediate neighborhood of the cell body. Certainly, those fibers that take a horizontal course from the sides of the Purkinje cells to the neighboring ones can only be explained, as axis cylinders. Another peculiarity of the axis cylinder is its round about course in the granular layer. Some of the axis cylinders which

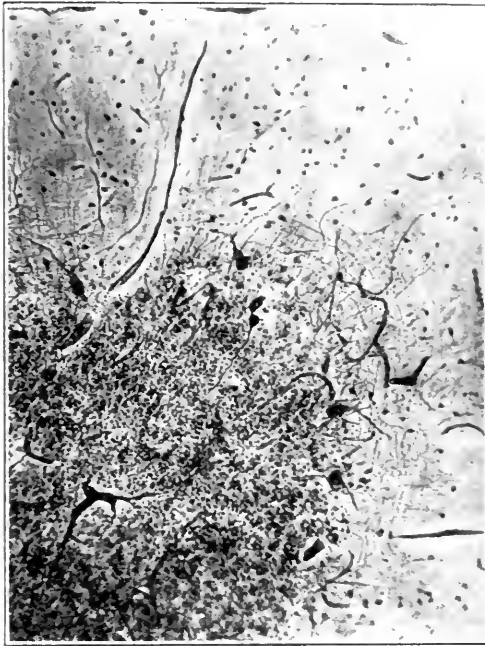


Fig. 2.—Senile cerebellum showing degeneration of dendrites, tangential fibers, Purkinje cells and spheroidal cells of the granular layer. The vessels are relatively increased.

enter the white substance do not do so directly, but extend backward in a horizontal direction for a fairly long distance, finally curving in the opposite direction and entering the white matter.

The axis cylinders of the Purkinje cells usually extend from the base of the cell body; occasionally they extend from the side of the cell. In atypical Purkinje cells of the molecular layer axis cylinders frequently extend, not only from the abnormal part of the cell body, but also from the dendrites, and sometimes from dendrites far from the cell body (Fig. 2).

The collaterals of the axis cylinders are usually given off far from the cell body at an acute angle and directed toward the molecular layer. They divide, while in the granular layer, into several branches, which help in the formation of the cushion fibers and also share in the formation of the basket fibers. The collaterals seem to terminate in the molecular layer. Purkinje cells, therefore, seem to stand in an intimate relationship to each other, either by means of their axis cylinders or their collaterals.

Basket Fibers of the Purkinje Cells.—As for the basket formation around the Purkinje cells, we found about the same condition as described by Schaffer. In addition to this we observed axis cylinders and their collaterals taking part in the formation of the baskets. In a few instances I have also seen axis cylinders of the spindle formed cells included in the formation of the baskets.

Ascending Fibers of the Molecular Layer.—Not only so-called climbing fibers but all fibers ascending the molecular layer tend to lean against or pass over the dendrites of the Purkinje cells. This has been asserted also by Schaffer. I have clearly seen collaterals and axis cylinders from Purkinje cells and large cells of the granular layer as well as tangential fibers climb along the apical dendrites of Purkinje cells for a considerable distance. The fibers, therefore, climbing along the dendrites are not always the "climbing fibers" of Cajal and Koelliker.

STUDY OF PATHOLOGIC CASES

The swelling of axis cylinders and dendrites, in various pathologic conditions, has been noted in the foregoing. I have selected forty-one cases in which all the patients died of psychoses. These forty-one cases have been grouped under nine different categories to determine, if possible, the relationship of these peculiar changes to the following groups of diseases: (1) senile dementia, (2) arteriosclerotic brain disease, (3) general paralysis, (4) congenital brain diseases, (5) dementia praecox, (6) manic depressive insanity, (7) alcoholic and toxic psychoses, (8) brain tumors and (9) myxedematous psychosis.

GROUP I. SENILE DEMENTIA

CASE 1 (Case No. 20719, Aut. No. 2114).—Female; psychosis of four years' standing; died at the age of 78 from bronchopneumonia.

CASE 2 (Case No. 20150, Aut. No. 2116).—Female; died at advanced age of lobar pneumonia.

CASE 3 (Case No. 19760, Aut. No. 2008).—Male; psychosis of two years' standing; died at the age of 72 of arteriosclerosis.

CASE 4 (Case No. 19817, Aut. No. 2035).—Male; psychosis of ten years' standing; died at the age of 97 of bronchopneumonia.

CASE 5 (Case No. 19922, Aut. No. 2036).—Female; psychosis of several months' standing; died at the age of 71 of arteriosclerosis.

CASE 6 (Case No. 20887, Aut. No. 2086).—Female; psychosis of five years' standing; died at the age of 65 of cardiorenal disease with hypostatic pneumonia.

CASE 7 (Case No. 20507, Aut. No. 2082).—Female; psychosis of eight years' standing; died at the age of 73 of metastatic tumor of the mediastinum.

CASE 8 (Case No. 16964, Aut. No. 1990).—Male; psychosis of ten months' standing; died at the age of 76 of arteriosclerosis and hypostatic pneumonia.

CASE 9 (Case No. 19981, Aut. No. 2003).—Female; psychosis of one years' standing; died at the age of 87 of chronic nephritis and mitral regurgitation.

CASE 10 (Case No. 16657, Aut. No. 1924).—Female; psychosis of five years' standing; died at the age of 76 of arteriosclerosis.

CASE 11 (Case No. 19202, Aut. No. 1932).—Male; psychosis of two years' standing; died at the age of 80 of chronic nephritis and chronic valvular disease.

CASE 12 (Case No. 20064, Aut. No. 2026).—Male; psychosis of two years' standing; died at the age of 89 of interstitial myocarditis.

CASE 13 (Case No. 19458, Aut. No. 2027).—Female; psychosis of several months' standing; died at the age of 77 of arteriosclerosis and bronchopneumonia.

CASE 14 (Case No. 17444, Aut. No. 2030).—Male; psychosis of nine years' standing; died at the age of 74 of gangrene of the left lower extremity.

CASE 15 (Case No. 20558, Aut. No. 2050).—Male; died at advanced years of coronary sclerosis.

CASE 16 (Case No. 18638, Aut. No. 1877).—Female; died at the age of 87 of chronic nephritis.

Pathologic Observations on Group 1.—All patients in Group 1 presented fairly abundant senile plaques with or without Alzheimer degeneration of neurofibrils. Some patients presented softening and hemorrhagic areas due to cerebral arteriosclerosis, but these latter were included in this group because they exhibited senile plaques and diffuse fatty degeneration of ganglion cells.

In interpreting peculiar changes of dendrites and axis cylinders described in this paper one must not lose sight of the general associated pathologic changes shown in these cerebella and their possible causative or resultant relationship. Hence a description of both the general and special changes will be given.

The pia mater, in most cases, was thickened and showed a considerable amount of pigment substance. The walls of the pial vessels were thickened and revealed a more or less advanced degenerative fatty change. The molecular layer was diminished in width, particularly at the summit of the folia. The tangential fibers were reduced in number. The basket cells, which give origin to tangential fibers, showed marked changes of disintegration and many had disappeared. Together with the disappearance of the greater part of the dendrites, which will be

described later, the above mentioned condition gives the architecture of the molecular layer a very simple plan. Vessels that showed more or less sclerotic changes, however, appeared to be increased, owing largely, I think, to a diminution of intervacular nervous elements. It is to be regarded only as a relative increase of the vessels and unlike the proliferative processes of vessels which Alzheimer and others pointed out as occurring in general paralysis and some other chronic degenerative processes of the brain. In senile dementia, at least in our cases, the regenerative and proliferative processes of the vessels have not been observed.

Amyloid corpuscles were, in general, increased, particularly in the uppermost and deepest portions of the molecular layer, while in the middle portion of this layer they were rarely encountered. These corpuscles were readily stained by Bielschowsky's method, and took silver diffusely.

In striking contrast to the diminution or disappearance of other nerve elements, that is, tangential fibers, dendrites, Purkinje cells, etc., the number of basket and cushion fibers surrounding the Purkinje cells were not much impaired. These fibers, chiefly those in the intermediate or Purkinje cell layer, even seemed to be increased. Where atrophic Purkinje cells with a few stumps of dendritic arborization remained and even where Purkinje cells had disappeared, these fibers, more particularly cushion fibers, showed enormous masses of tangled fibers. This condition was most markedly observed in cases 4, 9 and 12, (Plate 1, Fig. 1), the patients being 97, 87 and 89 years of age, respectively. Most of the basket and cushion fibers were derived, as explained before, from the so-called basket cells, and it is difficult to explain why these fibers appeared to be increased while the cells from which they originated degenerated and decreased in number. Whether or not these seemingly increased fibers are to be interpreted as a compensatory proliferation of fibers from some other source of origin or merely as a relative increase due to emaciation and disappearance of the Purkinje cells, is difficult to determine. Schaffer, in his study of pathologic changes in neurofibrils of the cerebellum, observed the disappearance of basket and cushion fibers in cases of amaurotic family idiocy; taboparalysis and senile dementia. In our study of sixteen cases of senile dementia, the opposite condition obtained.

In the intermediate layer, there were more or less numerous fat corpuscle cells, more abundant than found in any other part of the cerebellum. The granular layer showed a diminution in small spheroidal cells, and the whole layer appeared to be lighter than normal. This condition made it rather easy to follow the course of the axons and afferent fibers of the white substance.

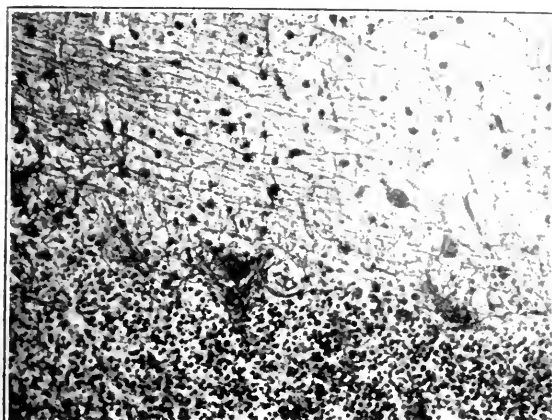


FIG. 1



FIG. 2

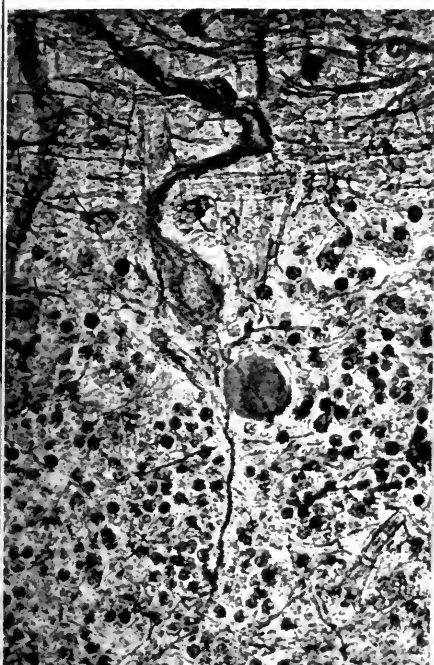


FIG. 3



FIG. 4

PLATE 1

Fig. 1.—Senile cerebellum, showing enormously increased cushion fibers.

Fig. 2.—Spindle formed swelling of an axis cylinder.

Fig. 3.—Pedunculated form of the axonal swelling.

Fig. 4.—Spherical swelling of an axis cylinder.

In the white substance, fat corpuscle cells were less numerous than in the Purkinje cell layer. In some cases a degeneration of myelin sheaths was observed, always accompanied by arteriosclerotic changes in the cortex or white matter.

In the cerebrum all cases of this group exhibited fairly abundant senile plaques and most of them showed typical so-called Alzheimer degeneration of the neurofibrils. In the cerebellum senile plaques were found only in Case 1. The Alzheimer degeneration, while abundant in the cerebrum, was not encountered in the Purkinje cells.

The Purkinje cell changes were of various kinds and were not always the same for each case of the group. The cells were more or less reduced in number, especially at the summit of the folia. The summit of the folia seemed to be the most vulnerable part of the cortex of the cerebellum. Of sixteen cases, eleven (Cases 3, 4, 5, 9, 10, 11, 12, 13, 15 and 16) showed perceptible diminution of cells, marked and universal in Cases 4, 13 and 15.

Most of the Purkinje cells were somewhat swollen and the Nissl bodies had partly or entirely disappeared. Some cells, however, were sclerotic, protoplasmic substance and Nissl bodies being stained dark. The protoplasmic prolongations in both of these cells were stained well and could be traced to a considerable distance. Cases 8, 12 and 16 showed extremely advanced fatty degeneration, while Cases 1, 2, 3, 4, 5, 9, 10, 11, 14 and 15 presented fairly marked fatty degeneration. This condition of fatty degeneration did not show any parallelism with the age of the patient or the stage of arteriosclerosis of the cortex. It is well to note that the fatty degeneration in the Purkinje cells in Case 1, which presented typical senile plaques in the cerebellum, was less marked than in the other cases which did not show senile plaques.

The most interesting findings in our study of the cerebellum were certain peculiar changes in axis cylinders and dendrites, which form the basis of this paper. I shall first describe the manifold changes of the axis cylinders and then those of the dendrites. There are roughly distinguishable two kinds of changes in the axis cylinders; a diffuse hypertrophy and localized swellings. A combination of these two may be encountered.

In the majority of cases of Group 1 the axis cylinders of the Purkinje cells were increased in thickness, not only the axonal stems but also the collateral branches. These hypertrophic axons were stained homogeneously dark; their neurofibrils were not visible. Associated with this was a diffuse disappearance of spheroidal cells of the granular layer which made it easy to determine the course of the axis cylinders. In this group were observed many instances of axis cylinders of Purkinje cells which did not enter the white substance of the

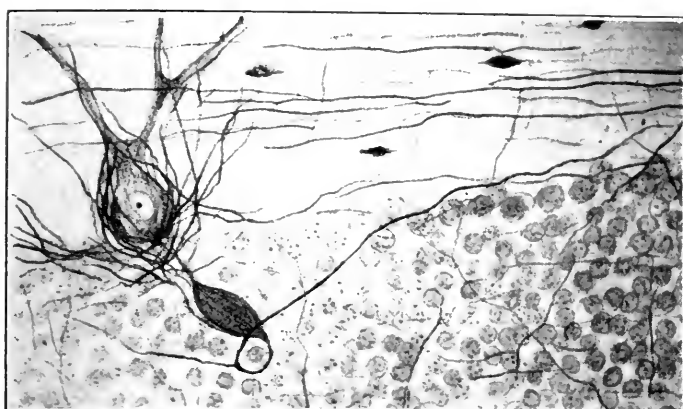


FIG. 1

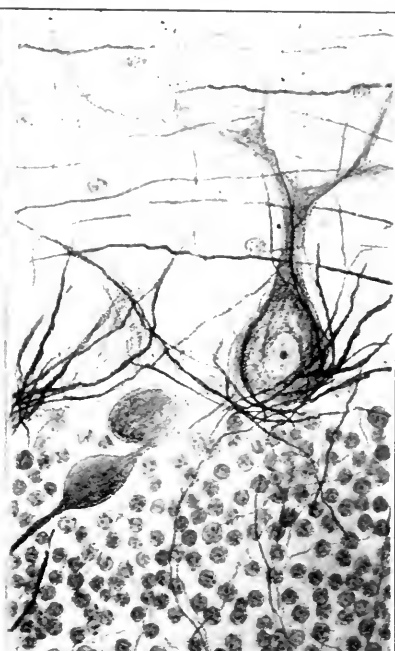


FIG. 2



FIG. 4

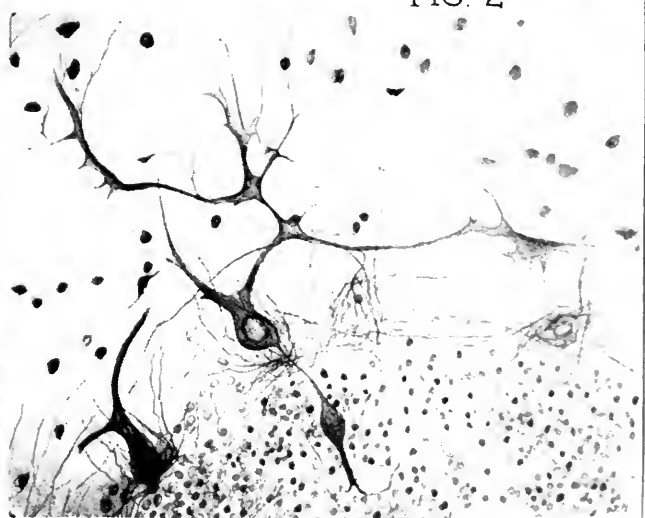


FIG. 3

PLATE 2

Fig. 1.—The axis cylinder presents a spindle formed swelling from which the axonal stem extends backward into the molecular layer.

Fig. 2.—A combination of pedunculated and spindle formed swellings of the axis cylinder.

Fig. 3.—Axonal and dendritic swellings of one and the same Purkinje cell.

Fig. 4.—Axonal and dendritic swellings.

cerebellum but which took their course to neighboring Purkinje cells, around which they arborized. This condition is normal, as mentioned in the consideration of the normal histology of the cerebellum, but in these cases it was decidedly noticeable because of the destruction of other nervous elements resulting in a simpler fiber pattern.

The localized swellings of axis cylinders which were identical with those described by Cajal, Rossi, Marinesco, Sträussler and Schaffer, were found in all the cases of senile dementia. They were most numerous in Cases 5, 6 and 11, numbering more than 50 in a small section (about 10 folia). The other cases, with the exception of Case 13,

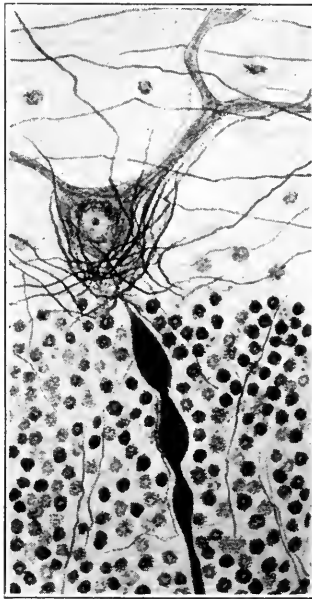


Fig. 3.—Beaded form of the axonal swelling.

exhibited from 5 to 20 swellings in a section of about the same size. In Case 13 only from 1 to 3 swellings were observed. Case 13 showed the most advanced cortical devastation, extreme fatty degeneration, marked disappearance of the Purkinje cells, considerable increase of amyloid corpuscles, etc. The histology of Case 5, which showed the most numerous examples of swellings, in contrast to Case 13, was comparatively normal, although individual Purkinje cells were more or less atrophic.

As a rule, Purkinje cells showing swellings of their axis cylinders are more or less atrophic, but not extremely degenerated. This perhaps explains why there were few axonal swellings in Case 13, in spite of the most marked degeneration of the cerebellar cortex, while in Case 5,

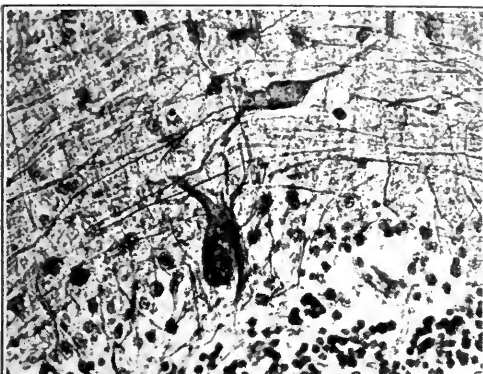


FIG. 1

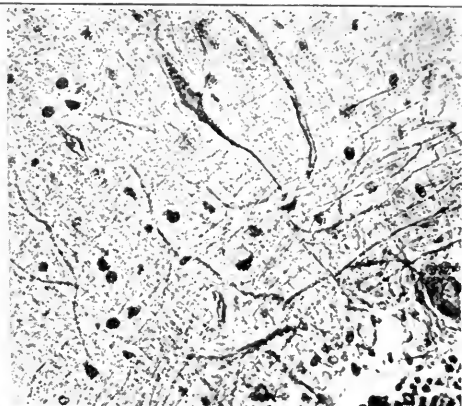


FIG. 2

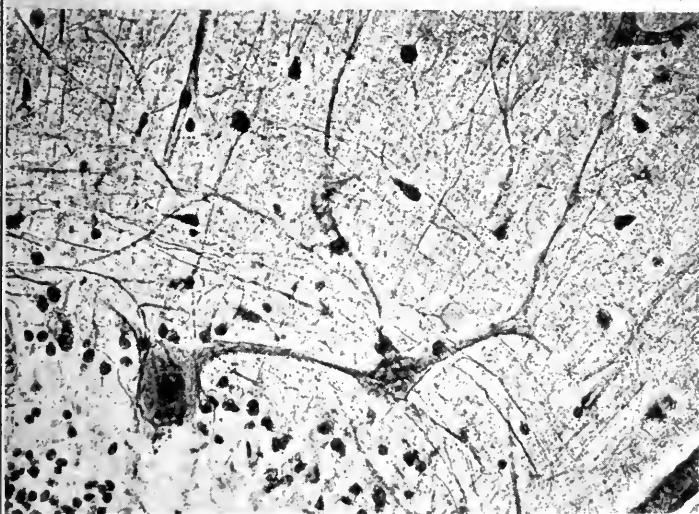


FIG. 3

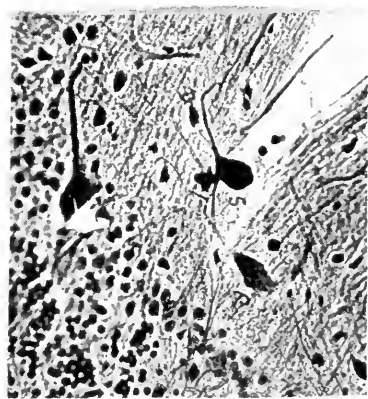


FIG. 4

PLATE 3

- Fig. 1.—Spindle formed dendritic swelling.
 Fig. 2.—Localized hypertrophy of a dendritic prolongation.
 Fig. 3.—Dendritic swelling of Type 4.
 Fig. 4.—Pedunculated form of a dendritic swelling of an atypical Purkinje cell; *P*, Purkinje cell body; *S*, swelling; *D*, dendrite.

which presented most abundant swellings, the Purkinje cells were only slightly degenerated.

Axonal swellings were usually located not far from the cell body, some in the immediate neighborhood of the latter, others a little farther away, in the granular layer, but always in the upper half of the granular layer. In Case 6 the writer observed a swelling of an axon coursing from a Purkinje cell horizontally to a neighboring Purkinje cell. When atypical Purkinje cells which are located in the molecular layer display swelling of their axons, swelling is likely to be found in the molecular layer. In no cases studied was swelling observed in the deeper part of the granular layer or in the white substance of the cerebellum.

The swelling is of various shapes: (1) the spindle form, the most commonly observed in all of the cases (Fig. 1, Plate 1, Fig. 2) (2) the



Fig. 4.—A peculiar type of axonal swelling resembling leaves of the cactus. Note also the transverse course of the axon.

conical form, less common, the apex of the cone turning either upward or downward (Fig. 2); (3) the spherical form, more rarely encountered (Plate 1, Fig. 4); (4) the beaded form, still more rarely observed, characterized by two or more spindle forms arranged like a string of beads (Fig. 2); (5) the pedunculated form, the rarest, characterized by hernia-like protrusion at the side of axonal stems (Plate 1, Fig. 3; Plate 2, Fig. 2; Plate 3, Fig. 4), and (6) the cactus like form, encountered in Case 6 (Fig. 4) in which an axis cylinder was swollen and from the swollen body another swelling issued, giving a resemblance to the leaves of the cactus.

Structure of the Swelling: The internal structure of the swollen body is not always the same. The great majority of the swollen bodies appear homogeneous, without any visible neurofibrillar structure. These homogeneous types are of two varieties, one staining pale, the other

dark (strongly argentophilic). A smaller number of the swelling bodies display definite intra-axonal neurofibrils; these, however, are pushed apart by some interneurofibrillar substance giving a loosely

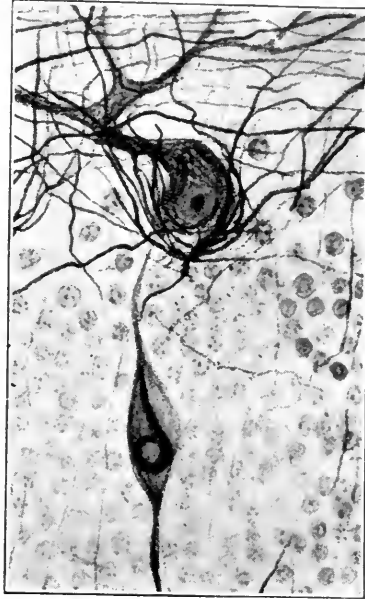


Fig. 5.—Alzheimer degeneration in the swelling of an axis cylinder.

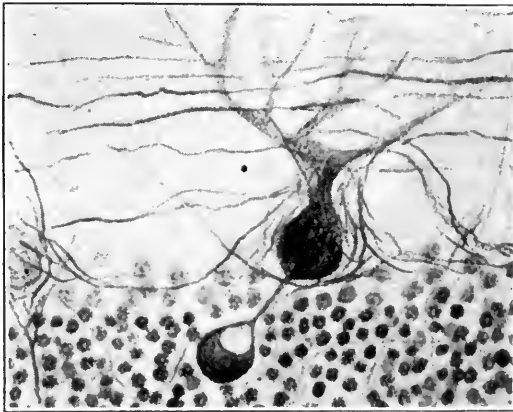


Fig. 6.—Vacuole formation in the swelling of an axis cylinder.

arranged appearance, the general direction of their course being unaltered. In a few cases I observed in one half of the swollen bodies a definite neurofibrillar structure and in the other half a more or less homogeneous argentophilic substance. I found in only two instances

thickening of neurofibrils and peculiar whirl-like structures suggesting Alzheimer degeneration of neurofibrils (Fig. 5). In the Purkinje cells, as mentioned before, no Alzheimer degeneration was found even after laborious search. This peculiar type of alteration found in two instances may possibly be of the same nature as Alzheimer degeneration. Other swollen bodies showed a coarse net formation, the interreticular substance being faintly stained. Still others displayed a dust-like substance in the swollen body, suggesting fragmentation of the intra-axonal neurofibrils. Vacuoles in the swelling of the axis cylinders, as shown in the illustrations of Marinesco's study, were not of

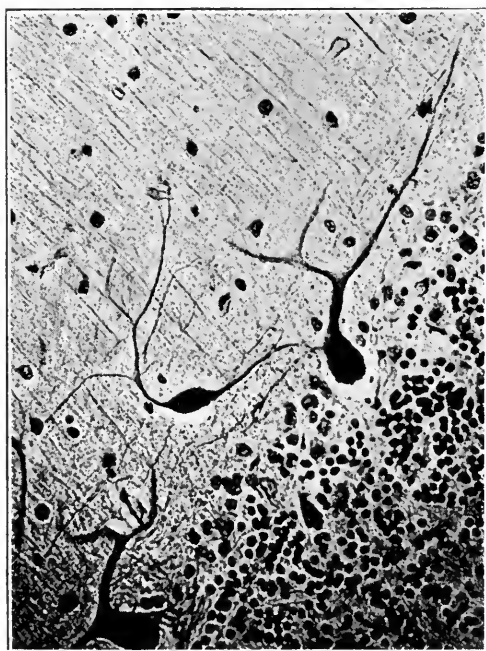


Fig. 7.—Spindle form swelling of a dendrite.

rare occurrence. Vacuoles were found mostly at the poles, rarely in the midportion of the swollen body (Fig. 6).

I tried to identify the substances in the peculiar swelling of axis cylinders by means of various methods of staining. In a small percentage lipoid substance, stained by sudan III and by the Marchi method, was demonstrated. The greater percentage of the swellings, however, showed no fatty content. A homogeneous substance with a glassy appearance was stained by the silver, in much the same manner as amyloid corpuscles, and some of these were markedly argentophilic. The exact nature of the substance is difficult to determine. Yet, the strong argentophilic character, the formation of vacuoles and

the deposition of fatty substance in some of them leads one to conclude that the processes are in part degenerative and in part regenerative, but that the latter are abortive.

Changes in the dendritic arborizations were not always alike for all the cases of this group. In Cases 1, 2, 6 and 8 the dendritic arborizations were well preserved, while in Cases 3, 7, 11 and 12 there was a partial disappearance of dendrites. Cases 4, 5, 9, 10, 13, 14,



Fig. 8.—Pedunculated form of the dendritic swelling.

15 and 16 presented a well marked degeneration and an extreme scarcity of dendritic prolongations (Fig. 2). In these cases, Purkinje cells possessed only stumps of primary and secondary branches; other finer branches had all disappeared.

The peculiar swelling of dendrites, which I believe that I am the first to describe in senile dementia, were found in nearly all cases of this group, namely: 1, 3, 4, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15 and 16.

fourteen out of sixteen cases studied, or 87.5 per cent. These peculiar dendritic changes were not found in equal intensity in all of the cases. Cases 4 and 9, especially, showed only a few. Where the process was slight the changes were more likely to be found in sections from the worm, probably because this portion of the cerebellum is the most vulnerable. Cases 2 and 12, which gave negative results for these changes, might have shown the same peculiarities if sections from more parts had been studied.

The types of dendritic swellings found were: 1. Spindle Form: (Plate 3, Fig. 1). These swellings were usually located distant from the cell body and were similar in appearance and structure to those found in axis cylinders of the Purkinje cells. This form might also be found in the main stems of the apical prolongation or in the secondary and tertiary branches but they were most frequently found in the dendrites given off from the side of the cell body (Fig. 7). Marinesco, in his paper on cerebellar regeneration, illustrated a similar condition, which he described as a pseudodendrite. I could arrive at no satisfactory explanation as to why those dendrites given off from the side of the cell body were more frequently affected than the normal dendrites given off from the apex of the cell.

2. Pedunculated Form: This type varied in size and was usually found on the stem of the primary, secondary or tertiary branches. In Case 3, as illustrated in Figure 8, the main stem showed two peculiar sac-like swellings from which fibers were given off to terminate freely in the molecular layer. These fibers might possibly have been dendritic arborizations, but unlike them they took a more or less serpentine course.

3. Bulb-Like Form: This type was always found at the end of a fiber where it has much the appearance of an electric light bulb (Fig. 9).

4. The next type of swelling was more or less spindle shaped and was always found at the points of branching into finer dendrites (Plate 3, Fig. 3).

5. This type displayed enormously thickened dendritic stumps from which radiated a great number of fibers, which formed a more or less tangled mass (Figs. 10 and 13).

6. This type consisted of a diffuse thickening of primary and secondary branches (Plate 3, Fig. 2).

The contents of the swollen parts were as varied as the contents in the swellings of the axis cylinders. The spindle formed swellings usually contained a markedly argentophilic homogeneous substance and resembled similar swellings in the axis cylinders. Pedunculated swellings were either homogeneous glassy matter which in staining quality

is very much like amyloid. This homogeneous glassy matter was probably a semifluid metabolic substance, but this could not be determined. The diffusely hypertrophic dendrites usually had a neurofibril content. Some, however, stained homogeneously. Other swellings contained vacuoles, as in the case of axis cylinders. A certain number of these swellings presented fatty changes, which were readily stained by sudan III, Scharlach R. and the Marchi method, but in most instances I



Fig. 9.—Bulblike form of the dendritic swelling.

was unable to demonstrate any fatty substances. The strong argentophilic character, the homogeneous glassy appearance, the vacuole formation, etc., point, as in the case of axis cylinders, to their degenerative character. The cases presenting these peculiar swellings showed, in most instances, well marked dendritic disintegration, and their degenerative character.

But is this process only degenerative in character? I observed in many instances numerous small branches extending from the swollen body which were fusiform, sac form or irregular swellings of the den-

dritic stumps (Fig. 10). This and the diffuse hypertrophy of the dendrites would indicate the regenerative nature of the process, according to my interpretation. This will be discussed later.

GROUP II. ARTERIOSCLEROTIC BRAIN DISEASES

CASE 17 (Case No. 21348, Aut. No. 2118).—Male; psychosis of one months' standing; died at the age of 77 of arteriosclerosis and bronchopneumonia.

CASE 18 (Case No. 17300, Aut. No. 2115).—Female; psychosis of twelve years' standing; died at the age of 70 of arteriosclerosis.

CASE 19 (Case No. 21459, Aut. No. 2128).—Male; psychosis of four years' standing; died at the age of 92 of arteriosclerosis.

CASE 20 (Case No. 21142, Aut. No. 2103).—Female; psychosis of eight years' standing; died at the age of 48 of chronic vegetative endocarditis.

CASE 21 (Case No. 17376, Aut. No. 1915).—Female; psychosis of five years' standing; died at the age of 79 of profound cerebral hemorrhage.

Pathologic Observations on Group 2.—In the cerebrum, all cases showed areas of softening and hemorrhagic lesions. Cases 17 and 18 presented small hemorrhages in the white substance of the cerebellum, as well as in the pons. Case 21 exhibited a severe new intraventricular hemorrhage, which caused the death of the patient. The larger arteries of the cerebrum, as well as those of the cerebellum, were markedly sclerotic. This condition, however, was most profound in Case 21. Case 20, previously reported by the writer as an atypical form of arteriosclerotic brain devastation, presented a peculiar gross appearance quite like the moth-eaten condition (*état vermoulu*) of Pierre Marie. Histopathologically the latter revealed "spongy degeneration of the cortex," in addition to hemorrhagic areas and softenings. In none of these cases were senile plaques or Alzheimer degeneration demonstrated.

The general histopathologic findings of the cerebellum varied in each case, as the group is vascular in origin and the changes depend on the grade of vessel alteration and location of diseased arteries, in this particular respect differing from the cases of the senile dementia described in the preceding group.

The cerebellar pia of these cases was irregularly thickened and adherent to the cortex by brushlike glia fibers (Fig. 11). The glia fibers of the border were here and there enormously thickened, often times dipping down into the molecular layer in areas which were wedge-shaped, the base of the wedge directed to the periphery. In the areas of arteriosclerotic devastation, the entire molecular layer was remarkably reduced in width, measuring only from one-third to one-fourth of the normal thickness. Cells and nerve fibers were almost entirely destroyed. Scarcely any Purkinje cells or their dendrites were encountered. Glia fibers, especially Bergmann fibers, were greatly

increased throughout the molecular layer. Amyloid corpuscles were seen mostly in the meshes of proliferated glia fibers of the outer border and in the deepest part of molecular layer.

In the intermediate layer a great many fat corpuscle cells came to view when the process was relatively young.

The granular layer and the white substance suffered equally from arteriosclerotic degeneration, causing in the former the disappearance of spheroid cells and in the latter the degeneration of myelin sheaths.

The fatty degeneration of Purkinje cells was also focal, that is, well marked in the lesion or in the immediate neighborhood of the



Fig. 10.—A dendritic swelling from which a great number of finer branches are given off.

lesion, while in remote parts it was barely noticeable. In Case 19 fatty degeneration was general and in a considerably advanced stage, the patient being 92 years of age.

Case 20 presented a peculiar alteration of the cerebellum. The degeneration of the nervous element was so great that within the affected areas all parenchymatous elements had entirely disappeared, leaving only a framework (Fig. 11). In the molecular layer the Bergmann fibers which traversed it were almost the only constituents to be seen, the whole layer appearing like a slat fence. In the intermediate zone the Purkinje cells had almost disappeared. Glia cells

with large nuclei remained and marked the border of the molecular and granular layer. In the granular layer spheroidal cells, Cajal's star cells and other cells of parenchymatous nature had all disappeared. Glia cells and fibers formed a loose network. Here and there, where the devastation was still more complete, one could observe only a cystic cavity with no trace of supporting fibers.

Local swellings of axis cylinders, such as those described for the preceding group, were also encountered. These, however, were not so numerous as in the preceding group. In a small section (about ten folia) from two to five such swellings were found in Cases 17,



Fig. 11 (Case 20).—An atypical form of arteriosclerotic devastation of the cerebellum.

18, 20 and 21, and in Case 19 in from three to four sections of approximately the same size, only one or two swellings were seen. The last case showed an advanced fatty degeneration of the Purkinje cells. As a rule, however, swellings are not usually found associated with markedly degenerated Purkinje cells. Most of the swellings were found far from arteriosclerotic lesions in apparently healthy tissue, as was noticed by Cajal. I could not decide whether some of these swellings represented divided central ends of axons of Purkinje cells. On the other hand, I was able to follow, in a few instances, axis cylinders

with swollen bodies far down into the white substance of the cerebellum.

The association of swellings with relatively healthy Purkinje cells, their location at a distance from the lesion, and the fact that swollen fibers can be followed farther down into the white matter, suggest a reaction in relatively healthy Purkinje cells and fibers.

In Case 17, as shown in Figure 12, a peculiar sort of swelling was observed—two spindle formed swellings connected by a fiber filament, one of them located in the intermediate layer where normal Purkinje cells were found and the other in the granular layer. The upper spindle showed a fine netlike structure, while the lower one presented a coarse network arrangement. Whether or not the upper swelling

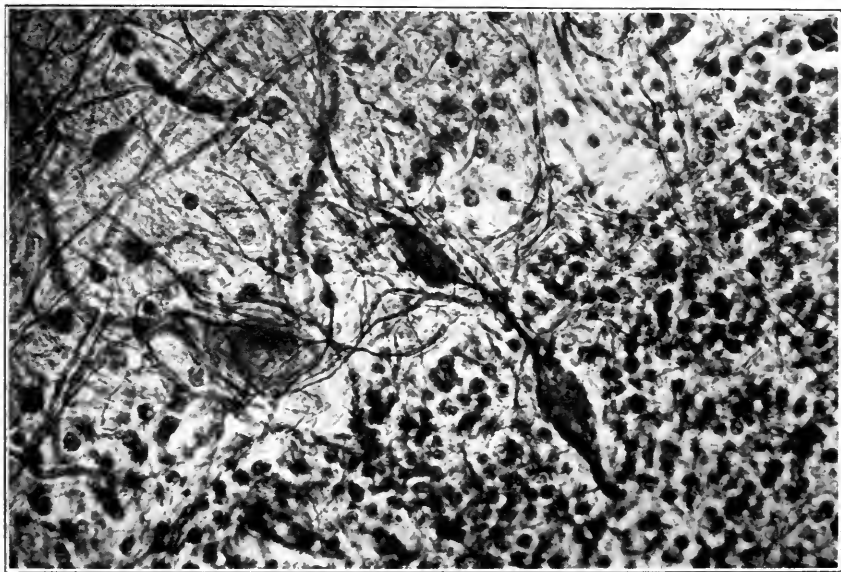


Fig. 12.—Photomicrograph of the cerebellum of the patient in Case 18.

was an atypical Purkinje cell or spindle formed ganglion cell, or a swelling of an apical prolongation from the spindle formed cell of the granular layer, is difficult to determine. The lower swelling might be either a local hypertrophy of a fiber, or it might be a cell body; but in any case, one of them was a structure of the type with which we are dealing. Swellings of fibers of unknown origin were found not only in the granular and the intermediate zones, but also in the molecular layer.

The dendrites of Purkinje cells were greatly affected within areas of lesions, but outside of them they were generally in fair condition. Case 21 presented a general scarcity of dendrites. The peculiar swellings of dendrites were found only in Case 21, and were fairly abun-

dant, being of many forms. A good example of Type 5 (described in the preceding group) was observed (Fig. 13). The ends of two dendrites were encountered which had the appearance of balls from which many very fine branches radiated. This condition suggested a regenerative rather than a degenerative process. The swollen or globular part was diffusely stained by silver. It did not contain any fatty substance. In other types of dendritic swellings a fatty substance

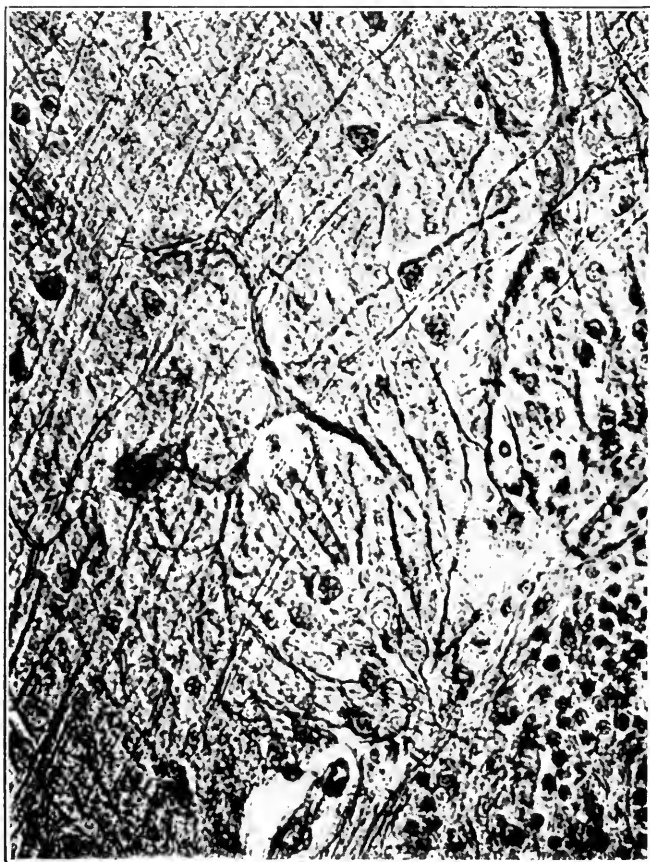


Fig. 13.—A type of dendritic swelling observed in Case 21. The ends of two dendrites show a ball-like swelling from which a number of finer branches are given off.

could be demonstrated, as well as a homogeneous semiliquid substance of an argentophilic character.

GROUP III. GENERAL PARALYSIS

CASE 22 (Case No. 19518, Aut. No. 1938).—Male; admitted at the age of 45; blood serum and spinal fluid positive; died two weeks after admission; cause of death was given as general paralysis.

CASE 23 (Case No. 18784, Aut. No. 1931). Male; admitted at the age of 48; fifteen years ago was infected with syphilis; blood serum and spinal fluid reactions positive. He died one year after admission to the hospital from lobar pneumonia.

CASE 24 (Case No. 18029, Aut. No. 1909).—Male; admitted at the age of 38; Wassermann reaction on blood serum and spinal fluid positive; died of bronchopneumonia after being in the hospital two years.

CASE 25 (Case No. 19395, Aut. No. 1924).—Male; admitted at the age of 39. He was admitted to this hospital eight years ago for the first time; at that time tests on spinal fluid were positive. He was admitted after eight years; died from general paralysis.

CASE 26 (Case No. 19091, Aut. No. 1971).—Female; admitted at the age of 51. Blood serum and spinal fluid reactions were positive; died one year after admission from general paralysis.

CASE 27 (Case No. 18681, Aut. No. 2030).—Male; admitted at the age of 60. Blood serum and spinal fluid reactions were positive; death at the age of 62 from general paralysis.

Pathologic Observations on Group 3.—The pia mater was thickened, its vessels being infiltrated by lymphocytes and plasma cells. The glia network of the outer border was considerably increased. The molecular layer was reduced in width, the structural plan being greatly altered. Tangential fibers and cushion fibers were diminished. The amyloid corpuscles were markedly increased; they were found, not only in the molecular, but also in the intermediate and granular, layer. Fat corpuscle cells were found around the infiltrated vessels in all layers. In the granular layer spheroidal cells were markedly diminished in number, especially in Cases 22 and 26. In the white substance there was a marked pallor of the myelin sheaths due to diffuse degeneration of fibers. Glia cells were increased in all layers, particularly at the outer border of the cortex, in the intermediate zone and in the granular layer. Glia fibers, as well as glia cells, were markedly increased, especially the Bergmann fibers.

The apical dendrites of the Purkinje cells tended to stain intensively and could be traced a greater distance than in the normal cerebellum. In Cases 22 and 26 the lipoid substance in the Purkinje cells was enormously increased; there was more of this substance even than was found in the most advanced cases of senile dementia. The Nissl bodies had disintegrated into a granular or dustlike substance. The nuclei of the Purkinje cells displayed various changes. They were mostly irregular in form and showed a dark stained nucleolus surrounded by a dark chromatin substance arranged in the form of a wreath.

All cases in this group displayed axonal swellings, but not so numerous as in the cases of senile dementia. These swellings were more numerous in Case 24. The character of the swollen bodies was the

same as that of the swollen bodies described in the cases of senile dementia. There was, however, less variety; most of the swellings were spindle or conical shaped.

Dendritic swellings were also observed in Case 22. In addition to localized swellings a few dendrites showed diffuse hypertrophy, with increased staining quality and with a loss of the neurofibrillary structure. The swellings of the dendrites, mostly spindle or bulb form, contained fatty substance, which was readily stained by Scharlach R. Not only the localized swellings of the dendrites, but also diffuse hypertrophic dendrites contained fat corpuscles. The dendrites in this case (Case 22) seem to have suffered more intensively from degenera-

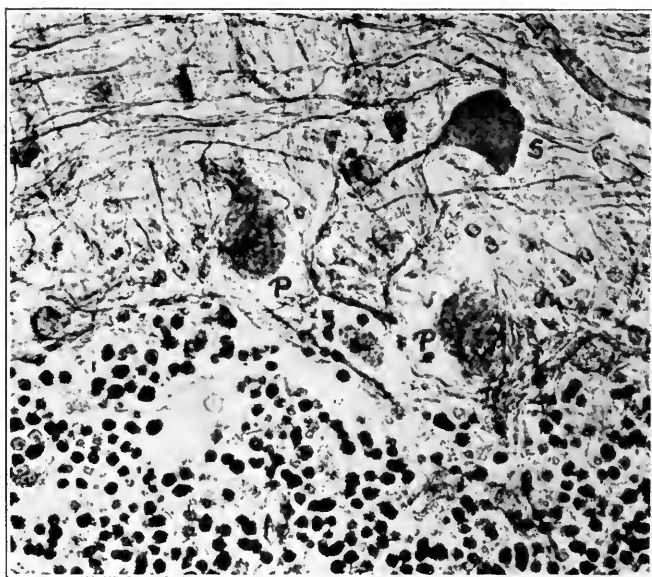


Fig. 14.—S, a swelling of a fiber whose origin is unknown; P, a Purkinje cell.

tion than in the remaining cases. Thus the swellings of dendrites appear to have a definite relationship to the general degenerative changes in dendrites. This may explain why dendritic swellings were found only in this case, for only in this case were general dendritic changes marked.

In Case 27 a few swellings of fibers were observed in the molecular layer in the immediate neighborhood of the Purkinje cells, but the cells of origin for these fibers were not determined. These swellings, however, may be fibers from spindle form ganglion cells of the granular layer, axons or collaterals of Purkinje cells, or dendrites given off from the sides of the Purkinje cell body, for I believe that any of these may show swellings of this sort (Fig. 14).

GROUP IV. CONGENITAL BRAIN DISEASES

CASE 28 (Case No. 16295, Aut. No. 2101).—Female; preceding the mental disturbance which occurred eighteen years ago, the patient had a "shock" following aphasia which lasted eight weeks. She died eighteen years after onset. Cause of death was given as chronic valvular disease.

CASE 29 (Case No. 19837, Aut. No. 2004).—Male; 8 years of age on admission; blind when admitted. He was untidy; he made no attempt to respond when questioned. He died eight months after admission; the cause of death was marasmus.

CASE 30 (Case No. 18551, Aut. No. 1999).—Male; admitted at the age of 62. The patient went to school but could not learn to read and write. He was classified as an imbecile. He died of tuberculosis.

Pathologic Observation on Group 4.—Case 28 was previously reported by the writer as a case of diffuse cerebrospinal sclerosis. The brain was small, weighing only 870 gm., and was unusually firm in consistency. Histopathologically, a considerable increase of glia cells throughout the whole central nervous system was shown. Myelin sheaths in the centrum semi-ovale were diffusely degenerated. In addition to these remarkable changes, there was a cyst in the anterior part of the centrum semi-ovale of the right side, surrounded by softened areas.

The cerebellum presented also a considerable increase of glia elements in all cortical layers and in the white matter. The nuclei of glia cells displayed all possible varieties. In the Purkinje cell layer abnormally large, irregularly shaped, nuclei of glia cells were observed. Rod cells were abundantly encountered in the white matter. Glia fibers also were increased. The glia belt of the outer border was enormously thickened. The Bergmann fibers were prominent. Heterotopic cells, from two to three in a few sections, were observed in the upper portion of the molecular layer.

I include this case within the congenital group on account of the abnormally small brain, simple convoluted pattern, heterotopy of Purkinje cells and some anomalous organs of the body.

Case 29 showed microscopically no characteristic findings of amaurotic family idiocy, which would be expected from a reading of the history. Ganglion cells of the cerebrum showed well marked fatty degeneration. Dendrites of the ganglion cells, however, were not swollen. Glia cells were increased both in the cerebrum and cerebellum. The cause of the blindness was not determined, nor could we determine whether it was of central or peripheral origin. As there were a number of anomalies and malformations in the central nervous system, as well as in the body, this case was classified in the congenital group.

Case 30 did not show any chronic inflammatory processes of the meninges and brain substance. The brain was relatively small (1,050

gm.), and the gyri were of a simple pattern. The evidence which points to the congenital nature of the case is not so good as in the preceding two cases.

In Cases 28 and 29 tangential and cushion fibers showed some diminution. The Purkinje cells were perceptibly reduced in number. Apical prolongations stained unusually well, while Nissl bodies showed a granular disintegration and disappearance, especially in the peripheral and apical portions. With the exception of a slight diminution in the number of Purkinje cells, Case 30 showed no remarkable changes either in the cells or in the fibers.

All these cases showed axonal swellings. The first two cases displayed abundant examples of swelling, while the last case exhibited few swellings. Most of the swellings were of the spindle form; other types were rarely encountered.

Dendritic swellings were observed in the first two cases; they were abundant in Case 29 (Fig. 15) and less abundant in Case 28. Most of the spindle form swellings occurred in secondary or in tertiary branches. They were markedly argentophilic and showed no neurofibrils. In Case 29 the swellings were encountered chiefly in dendrites given off from the sides of the cell body or in one of the secondary branches of less thickness. This condition was also observed in some of the cases of the senile group.

GROUP V. DEMENTIA PRAECOX

CASE 31 (Case No. 19440, Aut. No. 2007).—Female; age on admission 40; hallucinated; indifferent, lost interest in her work; at times refused food and medicine; died of bronchopneumonia after being in the hospital about a year.

CASE 32 (Case No. 20069, Aut. No. 2112).—Female; admitted at the age of 32, with ideas of persecution; hallucinated; indifferent and untidy. She died at the age of 33. Cause of death was given as exhaustion.

CASE 33 (Case No. 20403, Aut. No. 2051).—Female; admitted at the age of 32; unclean and hallucinated; disoriented. The Wassermann reaction on the blood serum was positive; spinal fluid reaction was negative. She died of mitral disease at the age of 34.

Pathologic Observation on Group 5.—No remarkable histopathologic changes were observed in cases of this group. In Case 31 there was a slight diminution of Purkinje cells at the summit of the folia. The amyloid corpuscles, in comparison with the other two cases, were slightly more numerous at the outer border of the cortex and in the deeper part of the molecular layer. The Purkinje cells were apparently normal in appearance, the finer architecture well shown. No swellings of either axons or dendrites could be demonstrated in any of these cases.

GROUP VI. MANIC DEPRESSIVE CASES

CASE 34 (Case No. 18081, Aut. No. 1993).—Male; admitted at the age of 49. He had two attacks of depression. He was apprehensive and had a tendency toward suicide. Two and a half years after admission he died of septicemia.

CASE 35 (Case No. 20058, Aut. No. 2015).—Female; admitted at the age of 51; agitation, extremely restless and resistive; flight of ideas. She died from general streptococcic infection two weeks after admission.

CASE 36 (Case No. 19918, Aut. No. 2017).—Male; admitted at the age of 45; depressed. He died three months after admission. Cause of death was given as arteriosclerosis.

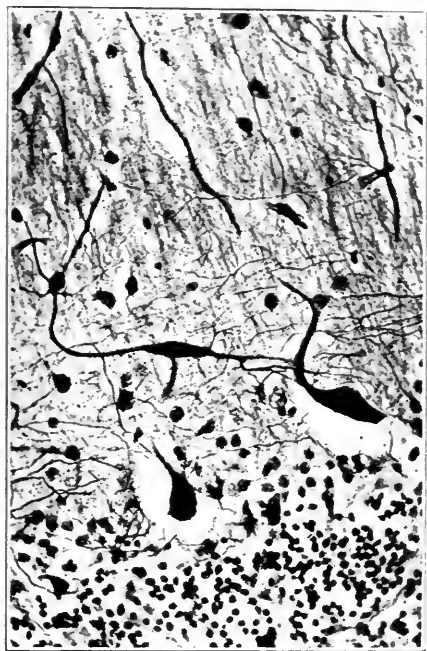


Fig. 15.—Spindle form swelling of a dendrite in Case 29.

Pathologic Observation on Group 6.—No remarkable histopathologic changes were found in these cases, with the exception of the changes in the Purkinje cells in the first two cases. In these cases a great many Purkinje cells had undergone a remarkable alteration. The nuclei, as well as protoplasm, stained poorly. The nuclear membrane had partly disappeared. The Nissl bodies were chromatolytic at the center, as well as at the periphery, of the cell. These changes of the Purkinje cells may possibly be associated with the terminal condition of the patients, who in both cases died of acute infectious diseases.

In none of these cases were the peculiar swellings of the axis cylinders and dendrites observed.

GROUP VII. ALCOHOLIC AND TOXIC CASES

CASE 37 (Case No. 9229, Aut. No. 1966).—Male; admitted at the age of 46 with a definite history of alcohol and attacks of delirium tremens. He died of lobar pneumonia after eighteen years in the hospital.

CASE 38 (Case No. 20036, Aut. No. 2067).—Male; admitted at the age of 50; definite history of alcohol. He died eleven months after admission of tuberculosis.

Pathologic Observation on Group 7.—The changes were marked and of the same character in both of these cases. Glia cells and glia fibers were slightly increased. The walls of the vessels displayed more or less fatty degeneration. Fat corpuscle cells were abundantly observed in the intermediate cell layer. The Purkinje cells were somewhat diminished in number, showing a marked increase of lipoid substance. Apical dendrites were rather deeply stained and could be traced for a considerable distance. The nuclei of the Purkinje cells showed some contraction and irregularity in form. The Nissl bodies had disintegrated. The tangential and cushion fibers seemed to be slightly diminished in number. Fat corpuscle cells were observed in the granular layer and in the white substance.

Swellings of the axis cylinders were observed in both cases, one or two in three or four sections. These were all spindle shaped and diffusely argentophilic.

Dendritic swellings were observed in Case 37. The swellings were spindle shaped or pedunculated. In Case 38 swellings of some fibers whose origins could not be identified, were found in the molecular layer not far from the Purkinje cells.

GROUP VIII. BRAIN TUMORS

CASE 39 (Case No. 21305, Aut. No. 2110).—Male; admitted at the age of 49; died ten days after admission.

CASE 40 (Case No. 20818, Aut. No. 2087).—Male; aged 50 on admission; aphasia; headaches with vomiting and vertigo.

In both cases the tumors were found to be gliomas. In both cases the cerebellum was flattened as a result of an increased intracranial pressure. Macroscopically, the cerebellar folia were generally flattened. Microscopically, the molecular layer was seen to be diminished in thickness, particularly at the summit of the folia, which gave them the appearance of a xiphoid process.

The most remarkable changes in this group were those in the dendrites of the Purkinje cells which coursed in a zigzag or serpentine manner (Fig. 16). Most of the finer dendritic branches had disappeared. The apical dendrites showed exceptionally good staining qualities. The Purkinje cells, basket and cushion fibers suffered very little. There was also a slight increase of glia cells and fibers.

The axis cylinders of the Purkinje cells disclosed the peculiar changes with which we are dealing. No dendritic swellings were observed.

GROUP IX. MYXEDEMATOUS PSYCHOSIS

CASE 41 (Case No. 21477, Aut. No. 2125).—Female; admitted at the age of 66. On admission she showed typical symptoms of myxedema. She died one month after admission. Cause of death given as myxedema.

Pathologic Observation on Group 9.—The ganglion cells of the cerebrum of this case showed cell changes due to a myxedematous condition of the brain matter. I have previously reported this case,

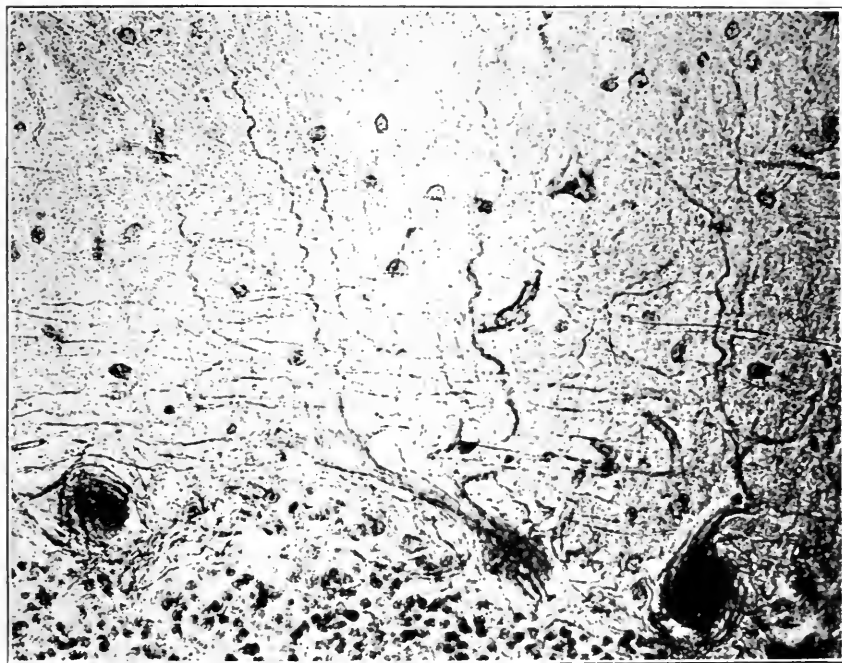


Fig. 16.—Serpentine course of dendrites due to pressure of the cerebellar cortex.

calling special attention to this type of cell change, which is believed to be pathognomonic for this disease. The Purkinje cells did not show this peculiar change. In the cerebellum the changes were very much like those of arteriosclerosis. The glia cells and fibers were increased. Fairly abundant amyloid corpuscle cells were found in the deeper portion of the molecular layer. The Purkinje cells contained only a moderate amount of fatty substances.

The peculiar swellings of axis cylinders, two or three in a section, were found. There were also some swollen fibers of obscure origin in the neighborhood of Purkinje cells.

SUMMARY AND CONCLUSIONS

The peculiar swellings of the axis cylinders and dendrites have been observed in various groups of diseases more commonly than was anticipated from a study of literature. These peculiar changes can, therefore, no longer be considered as specific changes. They are encountered in cerebella whenever there is a chronic degenerative process. This process may be either of an inflammatory character, the result of malformation, or of toxemia, as for example, from alcohol.

GROUPS OF CASES STUDIED, WITH THE PERCENTAGES OF AXONAL AND DENDRITIC SWELLINGS

Groups of Diseases	Cases Studied	Axonal Swellings	Percentage of Axonal Swellings	Dendritic Swellings	Percentage of Dendritic Swellings
Senile dementia.....	16	16	100	14	87.5
Arteriosclerosis.....	5	5	100	1	20.0
General paralysis.....	6	6	100	1	17.0
Congenital.....	3	3	100	2	66.7
Dementia praecox.....	3	0	0	0	0.0
Manic depressive.....	3	0	0	0	0.0
Alcohol, toxic.....	2	2	100	1	50.0
Brain tumors.....	2	2	100	0	0.0
Myxedema.....	1	1	100	0	0.0
Total.....	41	35	85	19	46.0

Axonal swellings are more frequently observed than dendritic swellings. Axonal swellings may be found in any disease in which the Purkinje cells have undergone degeneration. If, however, degeneration of the Purkinje cells is extreme, axonal swellings are either not to be observed or only a few are encountered as these swellings are stages of degeneration which these cells undergo. The dendritic swellings, on the other hand, are found even with fairly advanced degeneration of the finer dendritic processes and most commonly in senile dementia and congenital brain diseases. The dendritic and axonal swellings are not always of the same intensity in the same case though they are usually associated (Plate 2, Fig. 3).

What is the nature of these peculiar changes? Are they regenerative or degenerative in character? The swellings of the axis cylinders are not necessarily at the points of division of an axon by injury, although some swellings have no fiber continuing from them. From most of the swellings, however, a fiber may be followed down into the white matter. In cases which displayed coarse lesions, such as cystic cavities in the granular layer, the peculiar swellings were not found in the immediate neighborhood of the lesions but at a distance from the latter. Moreover, the swellings, as mentioned in the foregoing, have always been observed at a short distance from the Purkinje

cell body and always in the upper part of the granular layer. This fact and also the fact that the swellings are always associated with somewhat pathologic but not extremely degenerated Purkinje cells, lead to the assumption that the swellings are, in the beginning at least, a reactive process of pathologic but living protoplasm. The diffuse hypertrophy of axons and collaterals, which are found associated with focalized swellings, are to be regarded as a regenerative process. Thus the ill-nourished or slightly degenerating Purkinje cells appear to make a feeble attempt to increase the thickness of the axons. This increase of thickness may be, as Schaffer has asserted, the result of an hypertrophy of the axoplasm.

The "feeble attempt" at regeneration is, however, abortive. The Purkinje cells themselves, in diseases in which we find these peculiar changes, undergo degeneration, and their axons soon suffer a further process of a degenerative nature. This is expressed by the localized swellings of the axons caused by the accumulation of waste products of pathologic metabolism. The markedly argentophilic character of the swollen bodies, formation of vacuoles and deposition of fatty substances in the swellings, indicate the degenerative character of this phenomenon.

The dendritic swellings, though they are not so frequently observed as axonal swellings, probably are of the same character. Here I observed clearly that the dendritic stumps showed irregular swellings from which numerous finer branches were given off. This, and the diffuse thickness of the dendrites, leads one to conclude that the nature of the process is regenerative. Bulblike forms, which were found at the ends of thick dendritic fibers, may probably be compared to the central stump of the divided nerves, and are naturally of a regenerative character.

The regenerative process, however, is soon followed, as in the axis cylinders, by a degenerative one. The metabolic product which in most instances gives rise to the peculiar swellings is a homogeneously glassy substance. This substance shows a staining reaction similar to that of the amyloid bodies found in the central nervous system. I have been unable to determine the definite character of this substance. I have not yet arrived at a satisfactory explanation as to why senile dementia and congenital brain diseases are more likely to show this peculiar phenomenon than are other diseases. The inherited predisposition of the nerve element in congenital diseases and the acquired weakness in senile dementia may possibly play a great part.

REPEATED MULTIPLE MINUTE CORTICOSPINAL HEM-
ORRHAGES WITH MILIARY ANEURYSMS IN
A CASE OF ARTERIOSCLEROSIS *

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INTRODUCTORY

Miliary aneurysms were first mentioned by Cruveilhier¹ in his "Atlas," in which he pictured a case of multiple minute hemorrhages similar in pathologic appearance to the condition in the case I shall describe. He reported this condition as a "rare form of capillary apoplexy which consists of small disseminated miliary hemorrhagic foci (*apoplexie capillaire a foyers miliaries*). In 1851, Virchow² carefully described miliary aneurysms, and in his plates showed that true aneurysms are caused by disease in the coats of the vessels, chiefly the muscular. He differentiated between spindle-form aneurysms which involve the whole vessel wall and sac-like swellings which indicate a weakening on one side only; the latter are much less common. He was able to show all vessel coats extending over some of the ectasias, including the middle or muscular coat—this condition he calls "aneurysmata vera totalia." Furthermore, he showed all degrees of degeneration of the muscle wall in these aneurysms; in some, the muscle coat was entirely gone in spots, leaving only the inner and outer coats.

Calmeil,³ as quoted by Charcot and Bouchard, also noticed them, but did not comment on their nature.

Gull⁴ observed the condition and demonstrated a ruptured miliary aneurysm in a case with cerebral hemorrhage.

Charcot and Bouchard,⁵ in 1868, made careful studies in a series of eighty-four cases of cerebral hemorrhage and applied the term

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1. Cruveilhier: *Anatomie pathologique du corps humain*, 1835-1842, Liv. XXXIII, Pl. II, Fig. III.

2. Virchow: *Virchows Arch. f. path. Anat.* **3**:442, 1851, Plate IV.

3. Calmeil: *Traité de maladies inflammatoires du cerveau*, 1859, T. II, p. 522.

4. Cases of Aneurysm of the Cerebral Vessels, *Guy's Hospital Reports*, Sec. III, T. V., London, 1859.

5. Charcot and Bouchard: *De l'hémorrhagie cérébrale*, *Arch. de Phys.*, 1869, No. 1, p. 112.

"miliary aneurysms."⁶ These observers were the first to point out their frequency as a cause of cerebral hemorrhage. They asserted that miliary aneurysms are most common in the basal ganglia and pons, at which sites Meynert⁷ and Heschl⁸ had noticed them; the next site in order of frequency is the cortex. According to Charcot, they are rare in other parts of the brain.

Ford Robertson⁹ brought out the fact that miliary aneurysms are common in the pia mater: also that it was not true, as Charcot and Bouchard had stated, that all miliary aneurysms were visible by the unaided eye.

Pick¹⁰ showed, in a convincing way, the frequency, disposition and form of miliary aneurysms by his method of washing the gray matter in saline solution or water, leaving the vessel stalks with aneurysmal dilatations plainly visible. He examined, with Ellis,¹¹ about thirty cases. He stated, in his conclusions, that he was unable to demonstrate true miliary aneurysms either as circumscribed ectasias or sac-like forms involving only a part of the vessel circumference. His main conclusion from his examination was counter to the Charcot-Bouchard theory that miliary aneurysms are the cause of fatal cerebral hemorrhage in the great majority of cases. Pick stated that such hemorrhages were due to ruptures of supermiliary aneurysms. He further stated that "neither the dissecting types nor the false aneurysms cause death." He admitted that "miliary aneurysms may rupture and that theoretically the simultaneous rupture of many of them might cause death. But there is no evidence of this."

More recently miliary aneurysms have been studied by Shennan,¹² who, in one of his cases, was able to show the lenticulo-striate artery well bedecked with aneurysmal dilatations in the hemorrhagic hemisphere, and the same vessel remarkably free of ectasias on the non-hemorrhagic side. He also found large fusiform ectasias—larger than miliary—probably the supermiliary forms, whose rupture Pick is willing to admit as an adequate etiologic factor in fatal cerebral hemorrhage.

6. Cruveilhier, as mentioned before, had already used the designation "miliary."

7. Meynert: Ueber Gefässentartungen in der Varolsbrücke und den Gehirn Schenkeln, Allg. Wien. Wchnschr., 1864, 28.

8. Heschl: Die Capillar-Aneurysmen im Pons Varoli, Wien. med. Wchnschr., Sept. 6, 1865.

9. Robertson, Ford: Pathology of Mental Diseases, Edinburgh, 1900, p. 150.

10. Pick, L.: Ueber sogenannten miliaren Aneurysm der Hirngefässe, Berl. klin. Wchnschr. 47:325, 1910.

11. Ellis, A. G.: Pathogenesis of Spontaneous Cerebral Hemorrhage, Proc. Path. Soc., Philadelphia 12:197, 1909.

12. Shennan, T.: Miliary Aneurysms in Relation to Cerebral Hemorrhage, Edinburgh M. J. 15:245, 1915.

Opinion, therefore, is divided as to the degree in which military aneurysms are a factor in causation of lethal brain hemorrhage. Charcot and Bouchard asserted that a great majority of such deaths were due to military aneurysms. Pick said that few, if any, deaths were caused by ruptured military aneurysms.

We believe the following case supports the latter view, for there is evidence of repeated showers of military hemorrhages with recovery in each instance in spite of an extremely high blood pressure, except in the last instance, in which death was due to intercurrent disease.

REPORT OF A CASE

History.—Patient No. 18905, at the Boston State Hospital, was an Irish steamfitter, 59 years old at the time of his death. He had been regular in habits, a steady workman and supported his family adequately. He had used alcohol but not excessively; he had used none for the last ten years. He had always been well.

A transient convulsion in August, 1916, when 57 years of age, followed by residuals, ushered in the present trouble. Since that time, a progressive amnesia with repeated attacks of vertigo and seizures at irregular intervals from two weeks to three months had been evident. He complained frequently of headache. During the seizures his face was flushed and generalized clonus was the rule, but there was no frothing of saliva.

In March, 1917, he was treated in a hospital (having been taken there after a convulsion on the street) for "high blood pressure." On April 25, he awakened groaning with a "fearful headache." He became blind and was seized by a convulsion followed by unconsciousness lasting seven hours. Anopsia continued a few hours longer. By the end of the second day he had recovered but still suffered from a headache. On the third day, he was confused and hallucinated (heard lumber falling). He was disoriented, and observation at the Psychopathic Hospital was advised by his physician. He was admitted on April 27, 1917.

First Physical Examination.—Examination of the chest and abdomen was negative, and the patient was well developed and well nourished. The blood pressure was: systolic, 230; diastolic, 160. Sclerosis of the superficial vessels and some speech defect focused the attention on the nervous and vascular system; and unequal knee jerks, a sluggish pupil (iridectomy in the other eye) together with a slight tremor of the tongue still further accentuated the interest. The urine examination was negative, and the renal function was only slightly impaired (50 per cent. in two hours, according to the phenolphthalein test). The spinal fluid contained globulin 1, albumin 2, cells 1; the colloidal gold curve was 3333332100, but the serum and fluid Wassermann tests for syphilis were negative.

First Mental Examination.—The patient was completely disoriented the first few days, but his mind became clear and he was then correctly oriented. His memory for recent events was defective, but he gave a fair account of his life, talked intelligently when not confused and had a fair grasp on surroundings. His personal appearance showed a careless attitude, though he probably had some insight into his condition. In ten days he went home improved, though still slightly confused and amnesic.

Second Admission to the Hospital.—He was returned to the hospital eighteen months later. During this time he had frequently been brought home by the police, being found in convulsions on the street; these occurred at almost biweekly intervals. One morning he awoke with numbness in the right arm and on the right side of the head. This numbness remained in the fingers after sensation had returned in the arm and face.

Second Physical Examination.—When examined at the Psychopathic Hospital, Oct. 24, 1918, he showed no physical change since the previous examination with the exception of a slight rise in blood pressure, which was systolic, 250 and diastolic, 160. On first admission the blood pressure was systolic, 230; diastolic, 160. The vessels were thickened and sclerotic as before, and the pulse full, regular and of high tension, though the urinalysis was negative, as were also the Wassermann and tuberculosis complement fixation tests of the blood serum. Nervous System: Pupils: There was coloboma in the left eye due to an old injury; the right eye was small and irregular and reacted in a small radius. Fundi: The retinal vessels were tortuous and showed considerable variation in size. The veins were deeply depressed and presented a silver wire appearance. Knee Jerks: The reflex on the left side was sluggish; the right reflex could not be obtained. The arm, abdominal and cremasteric reflexes were normal. There was no disturbance of the special senses or of the deep or cutaneous sensations; there were no tremors of the head, fingers or tongue, which protruded centrally.

Mental Status: When admitted the second time, the patient was quiet, pleasant and cooperative. He stated that it was 1873 (1918), and that he had never been at the hospital before. His wife stated that he had been irritable and unreasonable and had quarreled with all his relatives. He denied this in general, but admitted a "few arguments recently." His memory was poor and somewhat patchy, but better for the distant past; his judgment was defective. He attributed his present trouble to "high blood pressure," which "makes him irritable"; in this he showed partial insight. He displayed slight emotional exhalation, and though he had no delusions or hallucinations he seemed to have some difficulty in thinking and in expressing himself, but there was no motor speech defect.

Two days later he was oriented for place, shortly after talking freely and appearing to be happy, but that same night he became excited and smashed windows, sustaining several small glass cuts on hands and forearms. After this episode he was restless and confused and incoherent in explaining his conduct; he thought the police were pursuing him and that they must have wished to make a "damn fool of himself." The patient was exceedingly restless and noisy during the entire night and felt exhausted next morning. October 30 aphasia was noticed: Door was called "chair," the bandage on his hand a "spoon." He stated that he knew the articles but "cannot tell them." Later in the same day he became excited, rushed about the ward and became violent when attempts were made to control him. He had ideas of persecution and sought windows and doors in attempts to elude pursuers. The next day and for two weeks he was more quiet and rational but his mind was not entirely clear; then he had a convulsion of short duration. These convulsions occurred occasionally during the next six weeks, and he was in bed most of the time, took little interest in his surroundings and was markedly confused.

Jan. 25, 1919, he frequently sank into a lethargic state from which it was difficult to arouse him, though he was lying with his eyes open. He was aphasic and very amnesic. He had a convulsion lasting twenty minutes, followed by great confusion.

Two weeks later, Feb. 14, 1919, his temperature rose to 100 F., the next day it was 98.6 degrees. He was semicomatose; he could be aroused but soon reverted to lethargic state. Ten days later his right arm was spastic; it flexed at the elbow and wrist. Spasticity was present in the right leg. Knee jerks were absent as were also abdominal and cremasteric reflexes; and while there was no ankle clonus, there was a right-sided Babinski reflex.

March 6, 1919, the patient showed signs of circulatory retardation, both general and pulmonary. He remained comatose for four days and died on March 10.

Summary.—A man, aged 59 years, died after two years of untoward vascular symptoms, beginning with convulsions. He complained of headache; his relatives noticed irritability; and he was irregularly confused, disoriented, aphasic and amnesic. His blood pressure was high, from 230 to 250, systolic and 160, diastolic, but the heart and urine were normal. The reflexes gradually became unequal on the two sides, and evidences of brain involvement during the last six months presented themselves in increasing numbers of convulsions and confused periods, terminating in lethargy, coma and death.

Postmortem Examination.—The postmortem examination was made by Dr. M. M. Canavan fifteen hours after death. The body was that of a slender, poorly developed and nourished white man, 162 cm. in length. There were small superficial decubitus over the trochanters and sacrum. Rigor mortis was absent. The pupils were unequal; the diameter of the right pupil was 1 mm.; that of the left, 5 mm. There was coloboma of the left eye. The heart weighed 310 gm. The coronary vessels were prominent, sclerosed and constricted, but not occluded. The mitral and aortic valves were thickened. The heart muscle was firm. The lungs were congested; focal edema was present. No pneumonic processes were found. About 250 c.c. of sanguineous fluid were found in the left pleural cavity. The liver weighed 1,275 gm.; it was grayish-brown. The capsule was thickened. The lobules were distinct. The kidneys weighed 110 and 90 gm., respectively. The fibrous capsule was thickened and adherent. The pyramids were small and white. The cortex was grayish-white and 5 mm. deep. The vessels were thickened. Later microscopic examination showed marked hyaline degeneration of the tufts and small vessels. The lymph nodes at the head of the pancreas were enlarged. There was a duodenal ulceration 2 cm. by 0.8 cm. just below the pyloric ring. Marked sclerosis and tortuosity of the splenic artery were found. The aorta contained several medium-sized plaques of thickening, but not to a marked degree.

Calvarium, Brain and Cord: The inner table was smooth with the exception of depressions for paccchionian bodies and grooving for meningeal arteries. The dura was not adherent. The brain weighed 1,595 gm.

Brain: The second nerves were flat and slightly gray near the cerebral arteries. The third and fourth nerves were caught in thickened pia mater. The vessels were evenly white with yellow patches. There were fusiform aneurysmal dilatations of the vertebrals and of the posterior cerebellar arteries. The temporal, left frontal and hippocampal regions were liberally peppered with minute black and brownish-red circular areas, not larger than 1 mm. These were found to be minute hemorrhages. Near the pons on the right side was a linear cyst of softening, measuring 2 by 0.3 cm. The occipital pole on the right was flattened.

The superior surface of the brain showed slightly thickened dura and pia. The arachnoid was thickened along the vessels, which were themselves thick-

ened diffusely and focally. The same minute hemorrhagic spots appeared on the superior surface of the brain, particularly over the vertex. The convolutional pattern was fairly complex; there was scarcely any atrophy. The convolutions in the left hemisphere appeared to be somewhat flattened out and slightly larger than in the right hemisphere. The brain was firm throughout.

The Spinal Cord: This was softer and flatter than usual and at one point in the thoracic region, on section, a translucent area was seen surrounding the central canal (syringomyelia may have been present). In various sections small hemorrhages similar to those found in the cerebral hemispheres were plainly visible to the naked eye.

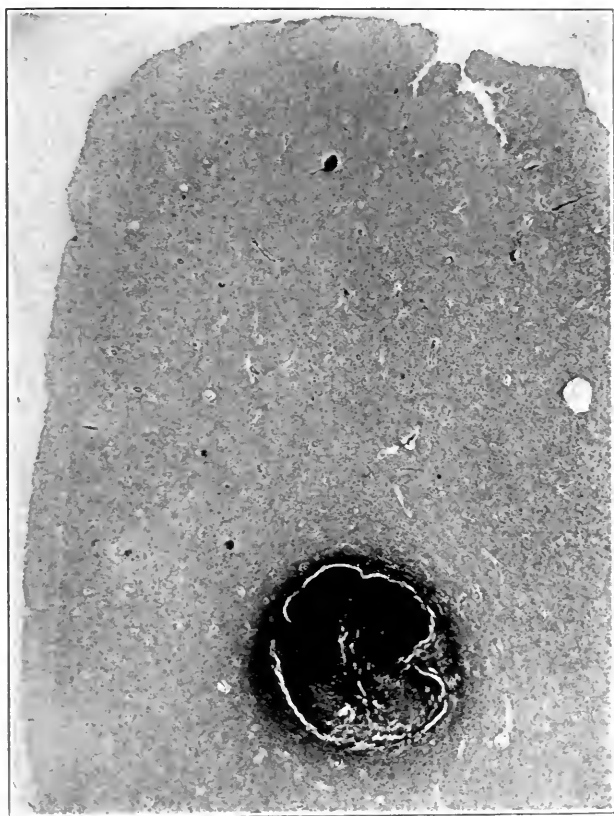


Fig. 1.—Recent hemorrhage from ruptured military aneurysm.

CROSS SECTIONS OF BRAIN

Over the convexity of the cerebral hemispheres numerous black spots from 1 mm. to 2 mm. in diameter showed evidence of recent hemorrhage. These areas stood out in even bolder contrast on the base, e. g., on the left hippocampal gyrus. In other instances the rupture and hemorrhage occurred more deeply in the cortex and was either faintly visible or entirely unseen from the surface.

These hemorrhagic areas could be divided into three classes; first the black, sharply demarcated spots; second, the less distinctly circumscribed faded yellowish-brown colored areas, and third, areas in which the brain substance appeared to be softened, less translucent and sometimes replaced by small cystic degenerations.

The first class included lesions of a recent date in which the blackish blood clot was sharply demarcated.

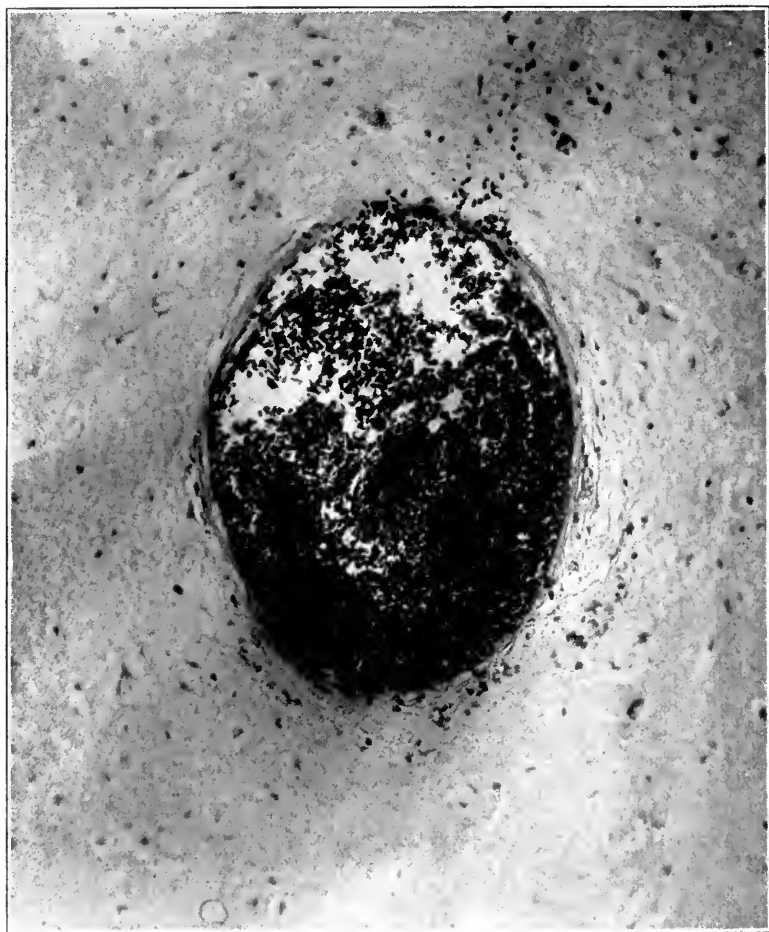


Fig. 2.—Cross section of an unruptured miliary aneurysm showing variation in loss of coats of vessel wall. Stained by Mallory's connective tissue stain.

In the second class were included the hemorrhages of longer standing, in which the disintegration of blood clot was shown in various progressive stages with fading and dissipation of blood pigment into neighboring tissue (Fig. 3).

The third class showed the end result. Pigment was no longer present, but in place of the areas of hemorrhage were found areas of softening, degeneration and, rarely, small cysts.

Most of the lesions were seen in the cortical gray matter but there were also some in the white matter. In the cerebellum the process was commonly in the white matter. In almost all these instances the hemorrhage occurred near either the internal or external surface, and rarely were the lesions deeply embedded. This may have been due to lessened resistance near the surfaces and greater support where the brain substance more completely surrounded the vessel.

EXAMINATION OF CORTICAL VESSELS

Blocks from various parts of the cortex were macerated in water, according to Pick's method.¹³ This brought most fruitful returns: the small vessels of the pia and branches dipping down into the cortex were found to contain numerous aneurysmal dilatations, fusiform, and sac-like; in some cases the small arteriole presented a moniliform appearance. It was found that in the hemorrhagic areas the aneurysms were innumerable. However, interval areas showed little aneurysmal formation and often several pial vessels were followed with no findings.

A favorite location was at the bifurcation of an arteriole, the entering vessel and one or both of the exit vessels showing irregular fusiform dilatations for a short distance. The size varied from 0.4 mm. to 0.9 or 1 mm., rarely larger; when the size was larger it was not much over 1 mm.

MICROSCOPIC EXAMINATION OF HEMORRHAGIC AREAS

The Recent Lesion.—Microscopic appearance of the various stages of resorption of the hemorrhages revealed the following: Vessels dilated and stretched beyond their normal caliber were present in the cortex in the neighborhood of small hemorrhages (Figs. 2 and 6). The amount of bleeding varied from the extravasation of a few cells to a small hematoma 1 mm. in diameter. In recent lesions hardly any reaction was visible. Gliosis was often present, not as a result of this, but probably on account of irritation due to malnutrition in the region of these thickened and athero-sclerotic vessels. By the Sharlach R. method fat, as a very early reaction, could readily be demonstrated in the area whose blood supply had been restricted. Later, blood pigment

13. The material had been hardened for several months, and in order to prepare it for this procedure, blocks were put in 1 per cent. sodium hydrate solution until softened—about three weeks. The vessels could then easily be separated and the branches dipping perpendicularly into the cortex from the pia would stand out stiffly showing clearly their relations.

appeared in abundance. The blood pigment was also frequently attached to ganglion cells and found free in the brain structure in large quantities.

The Old Lesion.—Destruction of the brain cell elements with neuroglia infiltration was seen in spots throughout the cortex. A lacy, open-work appearance showed the result of the previous lesion. Fragments of the vessel wall could still be seen, as shown by Van Gieson's



Fig. 3.—Site of old miliary hemorrhage showing pigment-laden phagocytes.

or Mallory's anilin blue stain, entwined by neuroglia fibrils. Active succulent glia cells were found in larger or smaller numbers in nearly all lesions. In myelin sheaths prepared by Weigert's method, a strand of degenerated fibers like the tail of a comet was often found in the wake of an old cortical hemorrhage (Fig. 6). It cannot be stated that all these lacy areas of gliosis were the end product of actual hemorrhage. An obliterative endarteritis seems to have been responsible, in

the cases of many of the smaller foci, for various degrees of endarteritic thickening; even obliterations without determinable hemorrhage were seen. Small cysts of softening no larger than 1.5 to 2 mm. were found several times in various parts of the brain.

Fenestrated Membrane (Verhoeff Stain).—In a shallow sulcus between two small gyri, a vessel 0.15 mm. in external diameter with thickened walls was seen, but the coats were readily differentiated. The fenestrated membrane stood out boldly and the nuclei of the muscle fibers in the media were plainly visible. Nearby was another vessel, 0.12 mm. in external diameter, and though the walls were about the same thickness, little differentiation was possible. The three coats were not distinct; while the adventitia could be located, the intima and media were fused: only now and then in the outer part of this fused layer small areas staining more lightly and of granular appearance seemed to indicate degenerated muscle nuclei. The fenestrated membrane was lost. The vessel walls stained more darkly than those of the more nearly normal vessel, and seemed to be composed of a hyaline substance which formed transverse marks (artefacts from cutting) which showed an inelastic quality, resisting the knife for a distance before yielding and producing a ribbed appearance like the cut surface of fresh bread. This dough-like consistency may account for the wide stretching of some of the vessels (Fig. 2). Judged by the number of layers of muscle fibers of which it was still possible to form some opinion by the number of degenerated muscle nuclei, this vessel should have a fenestrated membrane, for smaller vessels with only one layer of muscle fibers to the media distinctly showed the elastic membrane; but no trace of it was seen. It had undergone the same degeneration as the intima and media and had become lost in the fusion of the two. The adventitia also showed diminished nucleation.

In other instances¹⁴ the elastica appeared reduplicated as if free proliferation had occurred. In these cases the intima was also thickened but distinct. The media, however, was not so readily seen. It appeared to have been encroached on by the augmented elastica.

Summary of Microscopic Examination.—All evidence of a chronic arteriosclerosis of the larger and smaller vessels of the brain was found. The vessel changes varied in some instances, but in general seemed to be due to an extensive endarteritis which was in turn responsible for disease in the other coats, notably the muscularis, including the fenestrated membrane. The end results, destruction of brain tissue with gliosis, were seen in all parts of the brain.

Miliary aneurysms were found in foci scattered throughout the cortex. On section, no true "aneurysma totalia vera," such as Virchow²

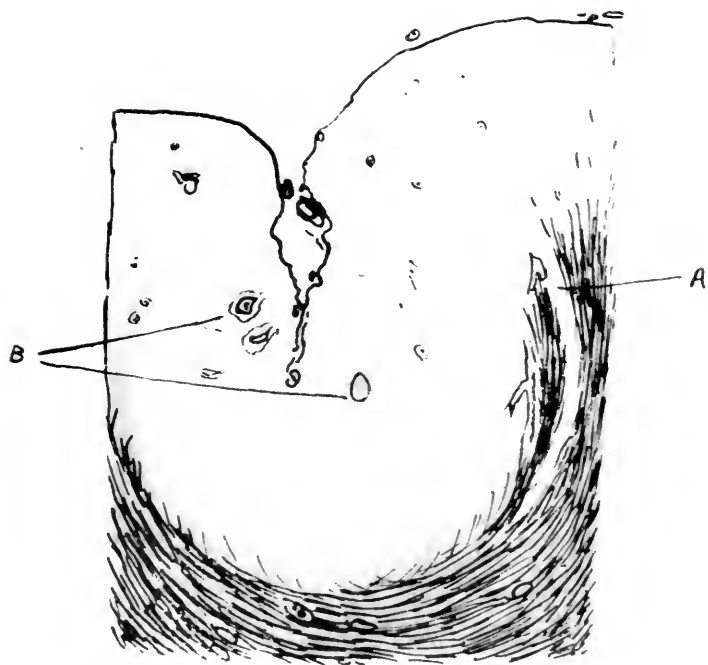
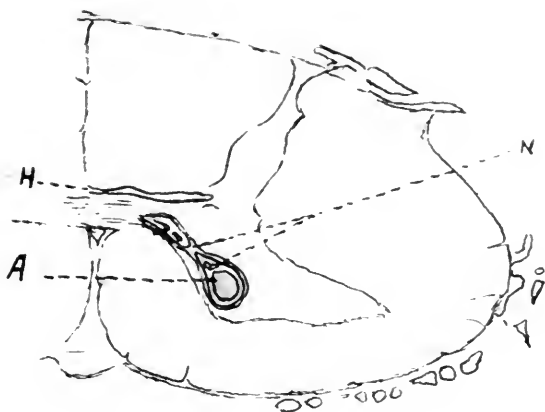
14. By Weigert's elastica stain.

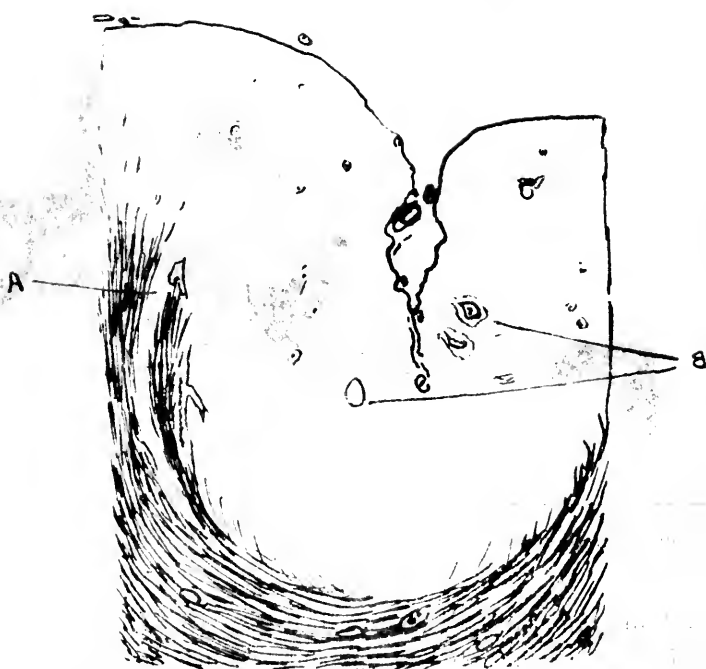
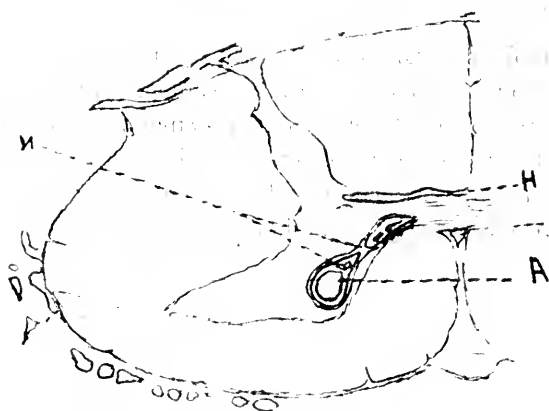
reported, with all the coats of the artery intact, was found. On the other hand, dissecting aneurysms were numerous—there were hundreds of them. False aneurysms due to local thickening of the coats were also seen, especially in the smaller vessels, but the dissecting variety was the most common. The intima was thickened and torn, the muscularis injured by fatty and later by granular degeneration,



Fig. 4.—Cortical aneurysms dissected out by Pick's method; unstained specimens: \times about 40.

and the elements of the blood had escaped into the intramural spaces, usually lifting the adventitia, thus forming the aneurysmal dilatation. On section, these aneurysms often appeared multilocular, some of the spaces being filled with a thrombus or fibrinous exudate. The lesions were also found in the spinal cord, but much more rarely. Here they





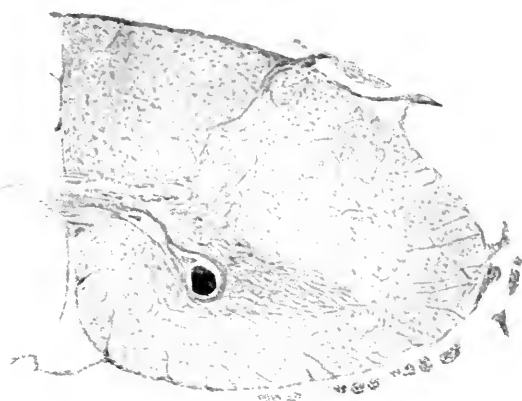
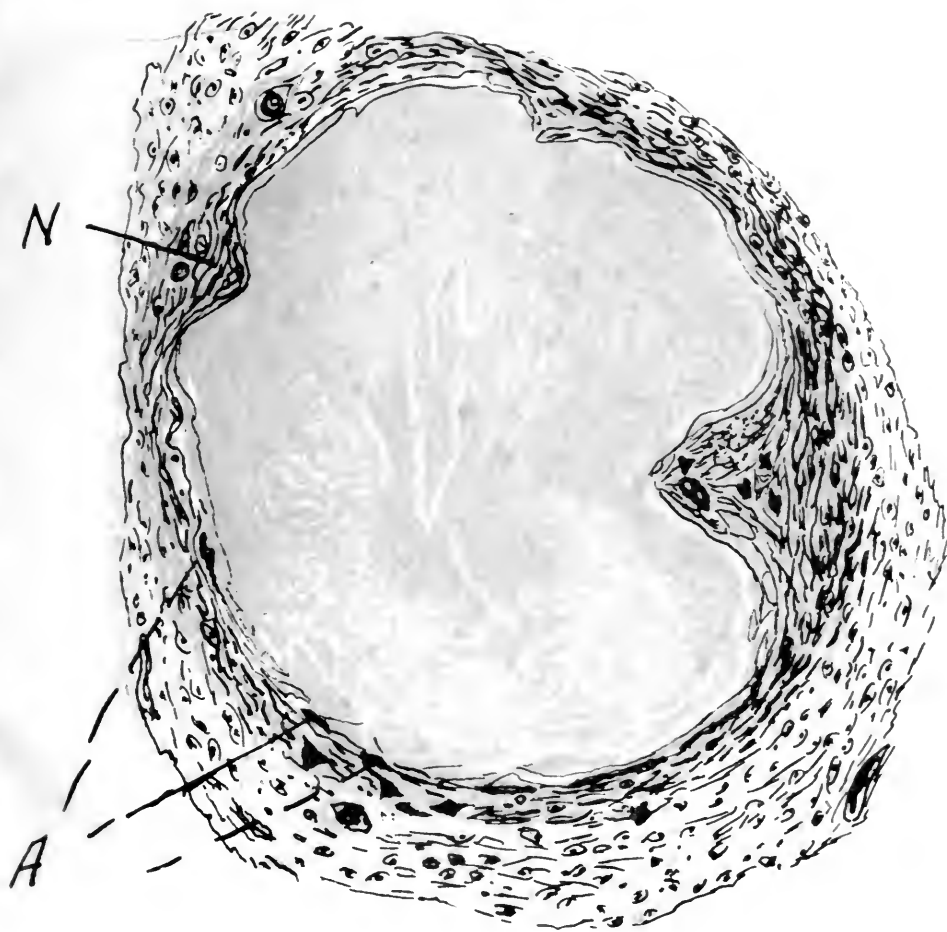
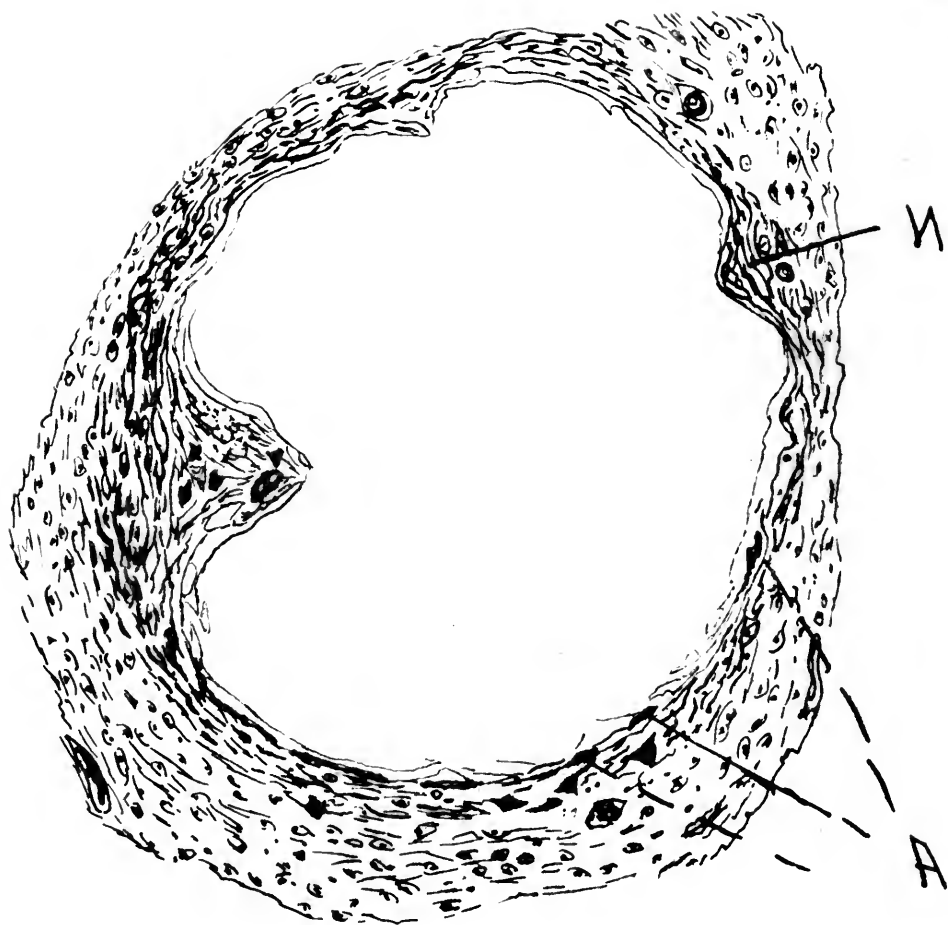


Fig. 5.—Section of lower cervical cord. Dissecting aneurysm at A with thrombus formation. A small channel near the periphery was still patent. N, neurogliosis surrounding dilated vessel; H, slight degree of hydromyelia. Mallory's phosphotungstic acid stain.



Fig. 6. Section of right postcentral gyrus; low magnification; n, Verloren stain. A, degenerated tract of fibers in wake of focal cortex lesion; B, thrombosed aneurysmal dilations.





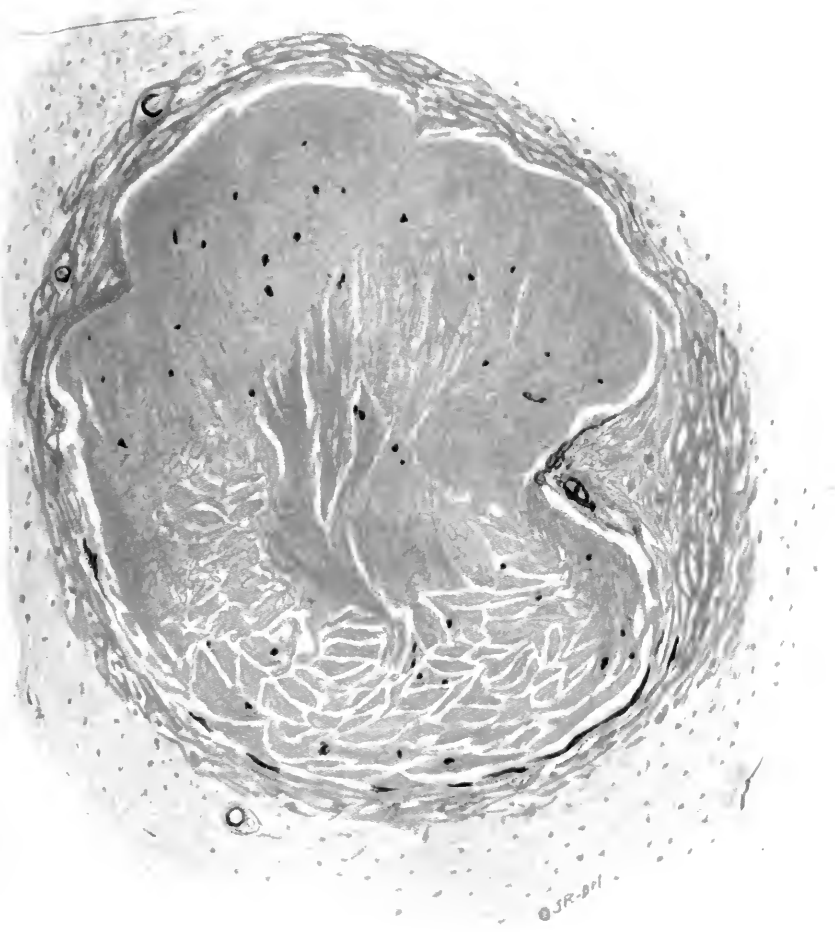


Fig. 7.—Hemorrhagic focus in left temporal region, showing fragments of connective tissue at A, the remains of a stretched and ruptured vessel wall scattered along the periphery on one side of the lesion. N, neuroglia, which have formed an artificial wall about the lesion which would make it appear on dissection like a true aneurysm. Pick found these false aneurysms in his cases. Mallory's anilin blue connective tissue stain; $\times 45$.

took the form of dissecting aneurysms surrounded by a dense layer of gliosis (Fig. 5).

No evidence of syphilis or lead intoxication, such as lymphocytosis or plasma cell infiltration, was found. A rod cell was occasionally seen. Active gliosis and fibrillar glial replacement of destroyed areas were general. Rarely, a small vessel showed infiltration with leukocytes, both polymorphonuclear and lymphocytic.

DISCUSSION

The clinical course of this case, with repeated convulsive attacks and confusion and other psychic manifestations, sometimes with aphasia, or amnesia or even paralysis, but always with rapid recovery, and a high blood pressure throughout, seemed to be fairly well correlated with the anatomic findings. The cause of the underlying vascular lesion was undetermined. So advanced a degree of arteritis in a man 57 years old is unusual. Alcohol, syphilis and lead are the first factors to be considered. The patient had used alcohol moderately; the Wassermann tests were negative; the patient had been a steamfitter, but had showed no sign of lead intoxication. There may have been a diathetic factor, though in the face of an occupation of a laborious and possibly toxic nature during a period of years, predisposition as a cause may have become a minor factor.

The histology of the brain showed evidence of advanced and long standing arteriosclerosis and hemorrhages of different age and degree. The small cysts and gliotic foci were indicative of old hemorrhages. According to the clinical history, the patient seemed to have suffered from such ruptures at various times in the last three years of life. Necropsy examination showed that there had been numerous small hemorrhages but despite the high blood pressure, no single large extravasation occurred. It seems that the advanced sclerosis and inelasticity of the finer ramifications of the vascular system might have been a protection against extensive hemorrhage from rupture of a small vessel or miliary aneurysm, since the force of the high pressure found in the large arterial trunks would have spent itself in overcoming the resistance of the stiffened finer tubules through which it was forced to pass. Therefore only a thinned out or diseased wall such as would obtain in a dissecting aneurysm, would predispose to rupture. In this case foci of various degrees of atherosclerosis were found. The condition of the finer arterioles varied greatly. When the disease had progressed to a certain stage in several foci in which the strength of the wall was reduced to a degree incompatible with the blood pressure conditions, an acute strain or unusual effort caused a shower of small hemorrhages. Figure 7 indicates the apparent steps of the process by

which these small hemorrhages occur. By a close examination of this lesions, both by the Van Giesen and Mallory-anilin-blue methods, one could distinguish fragments of connective tissue, i. e., of the degenerated wall stretched around the circumference of the lesion. These fragments, however, were found only in one part—practically confined to one half of the periphery. The vessel was probably stretched by aneurysmal dilatation, the walls, especially the muscular coat, was degenerated, the elastica was torn and finally, the wall was ruptured at the weakest point. The wide separation of the torn sides accounted for the absence of any fragments of vessel wall along that part of the periphery. It was possible for these small hemorrhages to occur slowly and to be arrested by the clotting of the escaped blood, though the positive clinical evidence pointed rather to sudden hemorrhaxis. During the final illness, in which minute hemorrhages occurred, death did not ensue until extensive pulmonary disease had developed.

CONCLUSIONS

1. In a case of marked arteriosclerosis with repeated convulsive attacks, evidence was found, especially in the cortex, of numerous minute hemorrhages of various age and size.

2. Miliary aneurysms, chiefly of the dissecting type, were found in large numbers—hundreds in the hemorrhagic foci. They were rare in the interfocal spaces. The various vessel coats could not all be followed over the aneurysms.

3. The vascular lesion was an arteritis, with degeneration of the fenestrated membrane and the muscularis, and frequently a degeneration of these coats resulted in a single layer of hyalin substance.

4. Repeated showers of small hemorrhages during a period of almost three years failed to cause death.

THE INTELLECTUAL STATUS OF PATIENTS WITH PARANOID DEMENTIA PRAECOX

ITS RELATION TO THE ORGANIC BRAIN CHANGES *

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The present investigation is an effort to obtain the intellectual status of apparently well preserved cases of paranoid dementia praecox. Gregor and Hänsel¹ hold that there is little impairment of memory until the terminal stages of the disease. Kraepelin² also considers that there is comparatively little involvement of memory until profound dementia sets in. In addition the latter finds that retention is often quite well preserved, while Gregor finds the power of retention considerably affected. It is in the field of memory and the higher associative processes that the present work is chiefly concerned, for it is felt that on the basis of the organic findings obtained by the author³ in her series of selected cases of dementia praecox an intellectual disintegration occurs much earlier than is generally conceded.

While the higher psychic processes are dependent on a preservation of memory, the ability to retain and recall perceptions which have been impressed on the brain cells at the height of their vigor and yet at the same time the inability to transform perceptions to general ideas and coordinate them into a logical whole shows a faulty psychic elaboration, a loosening of the unity and consistency of the psychic life, the basis of which may lie in the gradual disorganization of the connecting mechanisms of the various higher psychic centers.

Moreover, while volition and emotion influence the course of the psychic development, it is also true that they in turn are dependent on the intellectual activities, and a morbid process in the domain of the latter necessarily produces an emotional and volitional dulling with a resultant lack of intrapsychic coordination, a stoppage of further development and an ultimate disintegration in all fields of the mind as the disease process progresses, giving the characteristic picture of indifference to environmental influences with a psychology of everyday life which approaches those of races anthropologically lower.

That memory, the power of retention, logical association and constructive processes are as seriously affected as are the emotions and the will, seems evident from the results obtained. By memory is meant that form of independent psychic activity rather than the

1. Gregor and Hänsel: *Monatschr. f. Psychiatrie*, xxiii, 1.

2. Kraepelin, E.: *Psychiatrie*, Achte Auflage.

3. Rawlings, E.: *Histopathologic Findings in Dementia Praecox*, *Am. J. Insan.*, 1920.

mechanical memory which is shown in an acquired proficiency due to a long and continuous association of ideas and habits, which on close analysis approaches more nearly a reflex type of activity.

TABLE 1.—HISTORY OF PATIENTS TESTED

Case	Age	Schooling	Occupation	Duration of Psychosis in Years	Delusions	Hallucinations	Orientation	Use of Alcohol	Wassermann Reaction
2319	55	High school	Telegraph operator	14	+	+	+	Mod- erate	—
3456	35	11th grade, grammar school, business college	Bookkeeper	10	+	+	+	—	—
5025	38	High school	Structural engineer	5	+	+	+	Occa- sional	—
11358	33	High school graduate, college	Student	13	+	+	+	—	+++
15226	27	8th grade, grammar school, business college	Laborer	10	+	+	+	—	—
4014	64	Grammar school	None	44	±	±	+	—	—
10037	38	High school, business college	Bookkeeper	16	±	±	+	—	—
10425	34	High school graduate, law graduate	Admitted to bar	13	+	+	±	—	—
11710	49	University of Michigan, classical	Various	12	+	+	±	—	—
11830	43	9th grade, grammar school	School teacher	15	+	+	+	—	—
12249	36	8th grade, grammar school	Machinist	10	+	+	+	—	—
12993	36	High school graduate, law graduate	Failed to be admitted to bar	13	+	+	+	—	—
13542	52	8th grade, grammar school	Wood carver	24	+	+	+	—	—
14330	31	11th grade, grammar school	Machinist apprentice	12	+	+	+	—	—
15830	33	High school 2d year	Farmer	12	+	+	+	—	—
15903	42	High school graduate, college	Grover	15	+	+	+	—	—

The cases studied were carefully selected from among patients of native birth who had completed the grammar grade or received a higher education, and showed a fair or apparently normal intellectual preservation. An effort was made to select originally normal persons who had shown ability to utilize their scholastic opportunities, for it is well recognized that there are great individual differences in the original mental endowment which affect profoundly the capacity to profit from early training. Over fifty patients were tested, but on account of occasional delusional answers or evidences of inaccessibility or negativism, which made total scoring impossible, the list was narrowed down to sixteen. These cooperated in such a manner in all of the given tests that delusional trends and emotional and volitional aberrations had practically little or no influence on the thought processes. The patients were tested with the revised Yerkes-Bridges ⁴ point scale

4. Yerkes, R. M.; Bridges, J. W., and Hardwick, R. S.: A Point Scale for Measuring Mental Ability, Baltimore, Warwick & York, 1915.

in order to obtain the general level of intelligence. A series of other tests were given for the higher and more complex phases of mental activity, such as the power of comprehension, thought direction, abstraction, the ability to establish connections between concurrent and successive psychological activities, the capacity for response, etc., in order to obtain the intellectual deficiency, if any existed, along the lines in which they had been especially equipped.

Table 1 gives a summary of the important historical findings which may have a bearing on the present intellectual status of the patients.

INTELLIGENCE TESTS

Point Scale Tests.—In the point scale tests Professor Yerkes' revised graphs of 1917 were used in obtaining the norms. As all of the tests were made on men, the graph for males was used rather than the combined one. The results obtained showed a general average score of 72 points with a mental age of 10.9 years and an intellectual quotient of 68. Yerkes' twenty-five male mill operatives, with whom the patients may reasonably be compared, made a general average of 88.3 points; using the revised graph with its limits of 90 points and 16 years, they gave an average mental age of 14 years. In the tests, the subjects showed comparatively little of the scattering obtained in psychotic cases.

The analysis of the results shows that this series obtained a general average of 100 per cent. in the first three tests, 82 per cent. in the fourth or the memory test for digits, 100 per cent. in the fifth test, 46 per cent. in the sixth or the memory test for repeated sentences, rising again to 86 per cent. in the seventh test and then showing a rather progressive lowering in percentages obtained as the tests became approximately more difficult. It is interesting to note that they made such low averages in the memory tests, for they were apparently devoting their attention to the subject in hand and cooperating well in other respects. The results of the tests seem to demonstrate little involvement of the simple perceptual processes, sensory discrimination and esthetic judgment but considerable impairment of the processes of comparison, abstraction and generalization, a loss in the critical faculty and apperceptive activity. With one exception, the patients gave definite evidence of considerable intellectual deterioration. The results are shown in Table 2.

Kent-Rosanoff Test.—In the Kent-Rosanoff⁵ test for uncontrolled association the patients responded by a single term to each of a series of words. In this test they were found to be slower than normal and

5. Kent, Grace, and Rosanoff, A. J.: A Study of Association in Insanity, *Am. J. Insan.* 67:37, 390, 1910.

TABLE 2.—RESULTS WITH POINT SCALE TESTS

Case No.	Test																				Score	Mental Age	I. Q.
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20			
2319	3	4	3	3	4	4	2	6	8	3	4	1	0	6	2	3	6	6	6	81	11.9	0.73	
3456	3	4	3	3	4	4	2	2	4	1	2	2	4	8	0	2	6	1	2	60	9.0	0.56	
5025	3	4	3	3	4	4	2	2	8	2	2	4	4	6	4	4	5	2	0	72	11.0	0.68	
11358	3	4	3	5	4	2	8	0	4	8	3	4	2	2	6	2	3	4	0	72	11.2	0.70	
15226	3	4	3	4	4	2	9	2	8	0	3	1	2	2	4	2	2	4	0	62	9.2	0.57	
4014	3	4	3	3	4	2	9	2	7	2	4	6	0	8	4	4	4	5	4	75	11.2	0.70	
10037	3	4	3	4	4	2	6	2	4	3	3	0	0	4	1	1	0	2	0	51	8.4	0.52	
10425	3	4	3	5	4	2	7	2	5	3	3	0	0	7	1	4	1	2	0	61	10.6	0.66	
11710	3	4	3	5	4	4	4	3	6	3	3	1	4	7	4	3	4	6	2	81	11.8	0.73	
11830	3	4	3	3	4	2	9	1	8	3	4	1	4	6	3	4	4	2	4	86	12.2	0.76	
12249	3	4	3	3	4	2	8	2	4	4	2	4	1	4	4	2	0	4	4	64	9.6	0.60	
12963	3	4	3	4	4	3	3	2	8	3	0	3	0	4	2	0	4	6	2	60	9.0	0.56	
13542	3	4	3	4	4	0	3	2	8	3	0	3	4	1	0	4	2	4	6	75	11.2	0.70	
14330	3	4	3	4	4	4	8	2	6	7	3	4	1	4	8	1	3	6	6	83	11.8	0.73	
15830	3	4	3	5	4	4	9	2	7	3	3	2	3	4	6	1	3	4	0	75	11.2	0.70	
15903	3	4	3	5	4	6	9	2	5	2	2	4	1	4	8	4	5	6	4	94	16.0+	100.0	
Averages.....	3	4	3	4.1	4	2.8	7.8	1.7	4.7	6.7	2	3.4	1.3	2.2	5.6	2.2	2.7	4	2.5	4.3	72	10.9	68.0
Percentage of averages....	100	100	100	80	100	46	86	85	78	84	67	85	32	55	70	55	54	67	41	72	76.5	11.6	72.0

showed partial dissociations and preservations due to a moderate immobility of attention and an inability to dismiss from their minds previous stimulus words. Cases 10037 and 12993 showed a repetition of the stimulus words with multiverbal, nonspecific and predicative responses, the last type being especially numerous as their association times were unusually slow. The subjects seemed to grope for a response word more from a dearth or paucity of ideas than from the presence of emotional complexes. As a rule, their responses displayed more interest in the individual and concrete than in the more general and abstract, there seeming to be little interest in the far reaching relations existing between things. Their range of surface ideas was rather narrowed, and they were decidedly personal and subjective. The results of this test compared with the norms obtained in 1,000 adult cases by Kent and Rosanoff showed that these patients gave markedly fewer common associations, higher doubtful associations and strikingly increased individual associations, the last probably influenced by their delusional content. The test shows that they more nearly approached the norms for children under 11 years of age. It will be observed that the curve of performance in this test is somewhat parallel with that of the association test (No. 13) of the point scale, but it is more valuable in that it accentuates the deviation from the normal. The results are shown in Table 3.

TABLE 3.—RESULTS OF KENT-ROSANOFF UNCONTROLLED ASSOCIATION TEST

Case	Common	Doubtful	Individual	Failures
2319	60.5	7.8	25.2	6.5
3456	69.2	2.5	24.5	3.8
5025	70.6	3.4	24.5	1.5
11358	72.3	5.8	20.6	1.3
15226	60.5	3.5	30.2	5.8
4014	30.6	18.7	35.3	15.4
10037	28.2	15.5	38.0	18.3
10425	38.2	12.5	32.7	16.6
11710	62.3	4.6	27.1	6.0
11830	64.2	5.2	25.0	5.6
12249	60.0	2.8	29.2	8.0
12993	65.6	3.0	27.2	4.2
13542	72.3	8.0	15.4	4.3
14330	65.1	7.2	24.8	2.9
15830	75.5	3.5	21.0	0.0
15903	65.3	8.9	25.8	0.0
Averages	60.0	7.1	26.6	6.3
Kent-Rosanoff norms	91.7	1.5	6.8	0.0

Controlled Association Tests.—The controlled association tests, designed to discover the ability of the subject to appreciate logical relations and his power of adjustment to react according to instructions, were limited to the part-whole, the genius-species, the opposites tests and computations. In the part-whole tests the list of twenty words

recommended by Pyle ⁶ was used, for the genius-species test the Woodworth and Wells ⁷ twenty test words were used, and in the opposites test Pyle's twenty easy and hard opposites were used. The responses expected to the stimulus words in these tests were restricted to a single form of relationship throughout the series.

In computation, problems were given in addition, subtraction, multiplication and division. The recording of results was done at times by the patient, at others by the examiner, in order that it might be determined whether the process of recording disturbed his associations. The quality of the work was estimated as proportional to the percentage of correct solutions. Speed was not considered as valuable an index as the patient's mental environment during his hospital residence was less stimulating as to rapidity of mental processes than the environment of the normal person, and he was necessarily slower in time reactions. Moreover, individual differences in fatigability of the patients were so modified by the disease process that work curves could not be obtained which were of value as an index of original habit and method. The quantity of work was also modified by the same factors, the amount produced being much below the norms given for adults by various investigators. No attempt was made to correlate efficiency in the various forms of computation as the abilities demanded were too dependent on the original capacity of the patient to be estimated with any degree of accuracy.

The results obtained in these tests showed quite clearly that the patients were defective in their ability to reason, in their efforts to appreciate relationship and to control their associations. They fell within the range of children from 8 to 13 years of age, with one exception. The exceptional case patient, case 15903, ranged between 13 and 14 years, though he was shown to be normal by the Yerkes-Bridges tests. The total averages were: part-whole 8.3, genius-species 7.2, opposites 10.5. All of these were far below the norms for adults, according to Pyle, the part-whole average being 18.5, species-genius 15.1 and opposites 22.1. The general averages of these patients were those of Pyle's 11-year old children. In computations they made a general average of 64.8 per cent. of correct answers. Their responses were quite uniformly uninfluenced by their delusions, and they rarely failed to give an answer, cooperating readily and frequently with a child-like eagerness to do well. Their time of responses in the first three tests were so much longer than in the norms obtained by Woodworth and Wells and Pyle, and showed such variations that it was

6. Pyle, W. H.: *The Examination of Schoolchildren*, New York, 1913.

7. Woodworth, R. S., and Wells, F. L.: *Association Tests*, Psychol. Monog. **13**: 1910-1911.

decided to score solely in terms of number of correct associates without reference to time limit, as speed was considered of less importance than qualitative differences. The results are shown in Table 4.

TABLE 4.—THE CONTROLLED ASSOCIATION TEST

Case	Part-Whole Test	Genius-Species Test	Opposites Test	Computations of Correct Answers. Percentage
2319	8.5	7.8	11.0	55
3456	6.0	5.6	8.1	65
5025	7.8	7.2	10.6	70
11358	8.0	7.4	10.9	60
15226	6.3	6.0	8.6	38
4014	8.2	7.1	10.8	66
10087	6.1	5.1	8.0	50
10425	7.8	6.8	9.9	75
11710	8.9	7.9	11.2	88
11830	11.2	8.3	13.5	90
12249	7.1	6.1	9.0	48
12993	8.2	6.0	8.3	45
13542	8.5	7.5	11.0	69
14330	8.2	7.8	12.1	49
15830	8.9	7.5	11.2	72
15903	13.8	12.2	14.7	98
Averages	8.3	7.2	10.5	64.8
Pyle's norms	18.5	15.1	22.1	

TESTS OF IMAGINATION AND INVENTION

These tests were given to gage the patient's ability to think in images in the narrowest meaning of the term; that is, the presentation of their past experiences in such a new form that they did not refer definitely to any part of the same experiences. Every effort was made to break up the dreaming habits which many of the patients had formed and to direct their thought toward a purposeful effort to dissociate old combinations of past experiences and to combine them into active, creative productions. The individual differences in their mental traits in regard to wealth or paucity of spontaneous imagery in phantasy, and their capacity for creative thinking and ability to organize and plan, were shown to vary widely and to be quite generally less elastic and productive than would normally be expected of the average adult of common school education. While in a few instances their productions were colored by their delusional content, their train of thought could be sufficiently controlled to make the tests of value in judging their sense perceptions, their ability to form new associations and their fertility of imagination. As a rule, their qualitative classifications were decidedly commonplace, scientific and literary reminiscences being quite generally absent, even in those who had received more than a high school education. They showed their lack of fertility by frequently confining themselves to single types of imagery, which were nonconstructive in character. In order to allow their associations freer scope,

their responses were not written and their speed of associations was not estimated as it was found that they grew discouraged, their efforts at rapid associations seeming to retard their thought processes, limiting these frequently to objects within visual range.

Four tests were given: the ink blot test,⁸ the Masselon test,⁹ the development theme test⁸ and Ebbinghaus' completion test.¹⁰

The Ink Blot Test.—This test was used to find out the fertility of their visual imagination, the score being based on the total number of associations and the type and variety of the imagery, with no time limit. In this test four patients gave 8 associations, five gave 6, three gave 5, three gave 2 and one saw nothing in the ink spot, the average being 5.2 for associations. The type and variety of responses given were largely confined to the ordinary objects of their everyday life in the institution, few seeming to reach beyond it and then only along their delusional trends. The results of the test were a striking commentary on the nonconstructive ability of the patients as a whole, when it is considered that Pyle's adult males averaged 10.6 for associations with a time limit of three minutes.

Masselon's Test.—This test was elaborated into the invention story containing a number of prescribed words, the words being both nouns and verbs. Latitude was allowed in the use of singular or plural forms, possessive, nominative or objective cases in the nouns and any forms of the given verbs. In this test the responses were scored quantitatively as to number of sentences given and qualitatively as to whether the arrangement of the words was largely mechanical or whether they gave a concrete situation, and if given a concrete situation, whether it was dealt with in a limited or a fully outlined manner. The results in this test showed a correlation with the ink-blot test; the patients who manifested the greatest ability in the latter also showed the most constructive capacity in their development of sentences. They further showed that the sentences were largely mechanical in character, there being an awkwardness and inelasticity which suggested trains of thought that ran in ruts and which were switched along other lines to a great extent by delusional pressure.

Development Theme Test.—In this test the subjects chosen were mainly related to the patients' specific interests before the onset of their psychoses. As a rule, they were expository in type rather than imaginative, as it was felt that the imaginative type would give freer

8. Sharp, Stella E.: Individual Psychology, Am. J. Physiol., 1899.

9. Masselon, Psychologie des Déments précoces, 1904.

10. Ebbinghaus, H.: Ueber eine neue Methode zur Prüfung geistiger Fähigkeiten in ihrer Anwendung bei Schulkindern, Ztschr. f. Psychol. u. Physiol. d. Stimesorg. 13:401. 1897.

rein to the delusional trend and thus defeat the object in view. In this test the patients were given a time limit of fifteen minutes. Their productions were scored as to quality on a basis of the relative number of ideas elaborated and the judgment shown in the ideas expressed. The results of the test in the majority of cases were rather disappointing. With a few exceptions, there was a lack of constructive ability, and the ideas expressed were limited as a rule to commonplaces. Moreover, a generally low mental type was displayed, due to the absence of native retentiveness and capacity to recall.

Whipple¹¹ states that the relative number of ideas elaborated by the subject was indicated with fair approximation by the relative number of words written, so that the number of words may stand as a fair index of fluency of ideation and general linguistic readiness. Sharp's⁸ test subjects, in her study of university students, wrote an average of 259 words on imaginative and 222 on expository themes in ten minutes; her poorest subjects wrote an average of 124 and 94 words, respectively, for the same types of themes. The number of words produced by our group of patients ranged from 9 to 110, the average being 64 words. It is possible, to a certain extent, to compare their work with Sharp's poorest subjects, the averages of the latter being higher than in our series.

Results of Test: Several of the patients whose intellectual equipment best fitted them for this test responded thus: The patient in Case 5025, with the theme "Bridging the Niagara," produced a short sentence exposition as follows: "It can't be done. Yes, it could. I'd construct piers, eight of them and span it with cement. Sink the piers deep enough into the river bed. Might have the piers hollow, as probably stronger. Easy to do. Make them of steel. Then build a cement floor from pier to pier." Case 11358.—Theme: "The Death of Lincoln." "Lincoln was president. Seward was the secretary of state. The president and all the members of his cabinet were in the theater Ford in Washington. Booth did it." Case 10425.—Theme: "The Delays of Justice." "There are lots of delays in justice, due to one thing and another. Can't do anything about it. Just have to wait as trouble is often with the politicians. Sometimes you have to hunt up evidence to prove you have to hang a man or put him in jail and that takes time. Takes money to do things. Lot of delays in law." Case 11710.—Theme: "Who Influenced the World to a Greater Extent, Rome or Greece?" "The Spartans, they were from Sparta and the Athenians, they were from Athens and were as refined and cultured as could be. I suppose the Grecians influenced the most and still I don't know. I suppose the Romans. I guess the Grecians, but in world power I guess the Romans in war powers was a tremendous influence on the world in keeping the war pot boiling for years." Case 12993.—Theme: "Leniency of the Law Toward the Soldier." "Soldier fights. If he kills is supposed to kill." This was all that was obtained, although it was carefully explained to the patient that the leniency was in regard to general minor misdemeanors and not connected with

11. Whipple, G. M.: *Manual of Mental and Physical Tests*, Part II, *Complex Processes*, 1915.

the direct business of war, that of killing. Case 15903.—Theme: "Have Events Proven That the Sacrifices of the Civil War Justified the Freeing of the Negroes?" "Yes, for the reason that human liberty must be preserved and because slavery was degrading to both white men and black. The South is going to be more prosperous and more active because of better morals and competition. These things are providential, they are beyond our understanding sometimes." Case 2319.—Theme: "Death of a Dog." "A dog running after a wheeled cart was run down by an automobile. It is the duty of people to care for their dogs, especially when injured. The dog was badly injured and died immediately after and there was therefore nothing to do for him. He could have been taken to a hospital, a sort of dog hospital but as he was dead there was no use." Case 11830.—Theme: "The Influence of Newspapers." "The power of the press for forming public opinion can hardly be overestimated. Politics, daily tho't, our idea of the country at large all are shaped more or less by what we read on these subjects in the daily or weekly papers. Where yellow journalism does not overrule the factor of the newspaper is mainly for good. The births and deaths of our friends, is recorded there. The price of our commodities; the goings and comings of those we know. It ranks with the railroad and schools as a harbinger of civilization and educator of the masses. We could as well do without the church as without the newspapers."

Completion of the Test of Ebbinghaus.—Terman's eagle story, as given by Whipple, was used, entire words being elided. The patients were allowed to read the test and then fill in the elided spaces. They were given fifteen minutes and one trial only. Quality of work, not time, was considered. The results were computed on the basis of one credit for each elision filled in in any manner, 0.5 debit for each unfilled elision and one debit for each elision which did not make sense or for each word introduced in excess of the number called for; the second and third were subtracted from the first, and the percentage computed. As the test was considered by Ebbinghaus as especially valuable in gaging the intellectual activities that are fundamentally important and significant both in the school and in life, and as numerous other investigators have agreed in the main with him that it was a reliable test of intellectual ability, the results to be obtained with these patients were looked forward to with interest. Its performance required considerable diplomacy to obtain the cooperation of the patients as the elisions had a tendency to arouse delusional trends, in fact, for this reason a number of the patients were dropped from the list to be reported. The results obtained ranged between 20 and 65.8 per cent., the general average being 45 per cent. There was a rather marked reduction in each case, except in Case 15903, in percentage from the normal percentage to be expected from the original intellectual equipment of the patients, the results correlating with the rates obtained by the other tests. The lack of creative ability on the part of the patients as shown in their low general average, indicated fairly well the degree of intellectual deterioration which had taken place.

GENERAL AND SPECIFIC INFORMATION

Questions were asked in geography, history and arithmetic in order to establish a memory defect, if one existed. These questions were obtained by giving a number of general questions on these subjects to ten normal adults between the ages of 30 and 40, with an education not beyond the twelfth grade. From the questions answered correctly were selected five in geography, five in history and ten in arithmetic. The patients were also asked a number of questions on subjects studied in their college courses, care being taken to make them sufficiently simple to allow for the normal dulling of memory due to the lapse of time and the disuse of the knowledge acquired. The results showed that in only one case of the entire series of sixteen patients was the memory capacity for reproducing general and specific information at all equal to that of the normal person of like age or educational training. In all others the memory was seriously impaired.

SUMMARY AND CONCLUSIONS

The sixteen selected cases of paranoid dementia praecox scored by the Yerkes-Bridges point scale a general average of 76.5 points with a mental age of 11.6 and an intellectual quotient of 72. Various tests for uncontrolled and controlled association and for imagination and invention correlated rather well with the point scale scores, giving the patients a general average of 11 to 12 years. Tests for their general and specific knowledge demonstrated a diminution in linguistic fluency, patchy memories, ideation frequently commonplace and sterile in type, tendencies to deal with the concrete rather than the abstract when the latter was indicated, and a general crudeness and lack of flexibility in the use of their available mental equipment.

Correlating the clinical with the pathologic findings obtained, I found an impairment of the faculties of the mind, involving not only volition and emotion, but also the higher intellectual faculties of memory and the power of reasoning and the acquired capabilities. The disease process as a rule began insidiously with a progressive lowering of the intellectual levels, producing a gradual loosening of the train of thought with a resultant incoherence and disconnection, a deterioration in judgment, as shown in the patient's inability to order his life consistently from an environmental standpoint and the presence of more or less automatic or reflex activities which took the place of conscious purposeful action based on an intact psychic mechanism. Anatomically the destructive processes, while exerting their maximum effect on the frontal and central regions, diffusely attacked the cortex, most seriously affecting the more superficial nerve cell layers. These, so far as we are able to determine, are concerned with the phenomena

of memory and its associative mechanisms, the processes of abstraction which form the psychic personality, and the associating activity which takes place with the deeper cortical layers involved in volitional impulses and sensory perception.

Ariëns Kappers,¹² in his comparative work on the cortex, drew the conclusions that the neocortex, as distinct from the superficial layer of fibers, must consist of two functionally different zones: an outer supragranular stratum which was associative, receptive and sensitive in function and an inner and infragranular stratum which was corticofugal and commissural; between these two zones, in the granulated cortical regions, lies the granular cell layer, lamina granularis interna, whose short fibers he considers as intrahemispherical and short associative in character. He includes this lamina granularis interna with the two external receptive strata, making them all receptive and associative, the only difference being that the granular stratum establishes intracortical connections at short distances and the stratum above it at much longer distances.

E. G. Van't Hoog,¹³ in his study of the cortical strata and their functions, ventured the following conclusions: The cells of the lamina granularis interna should be considered as matrix cells which may become differentiated into the more highly differentiated supragranular pyramids and that animals whose neocortex is without this granular layer, or so sparsely supplied with it that they may be considered to be without it, belong to a class that has reached a blind alley in its development; the reserve cells are entirely expotentialized.

In the brains of my patients with dementia praecox I noted in the conclusions that there was a "singular fragmentation of the stellate nerve cell stratum in all the cases." It is this stratum that corresponds to Kapper's lamina granularis interna. In no other psychosis have I observed such a marked destruction of the nerve cells of the stellate layer of the brain, except possibly in the extremely atrophic senile cortex. Kappers' idea of the short associative character of this layer and Van't Hoog's premisis that it may be considered a matrix cell layer capable of differentiation into the cells of the upper strata throw a possible light on the peculiar rapid mental bankruptcy which occurs in the praecox psychosis. In view of Van't Hoog's conclusions, may we not be dealing in dementia praecox, with an embryonic condition in which the granular space is less than normal or of such lowered

12. Kappers, Ariën: The Phylogeneses of the Palaeocortex and Archicortex. Compared with the Evolution of the Visual Neo-Cortex, *Arch. of Neurol. & Psychiat.*, 1909.

13. Van't Hoog, E. G.: On Deep Localization in the Cerebral Cortex, *Trans. by Sylvia Jelliffe, J. Nerv. & Ment. Dis.* **51**:313 (April) 1920.

potentiality that the cells are incapable of differentiating into a normal type, or into a normal type beyond a certain period of life when they become exhausted under the stress of environment, or a possible toxemia due to a maladjustment of bodily functions? In the gradual destruction or exhaustion of this cell layer, we have the beginning break in the connecting fibers with the lower corticofugal cells with a disturbance of the sensorimotor reflexes producing the emotional and volitional aberrations observed in the disease. Moreover, as these cells become exhausted, we have a lack of the potential factors which make up the supragranular stratum, resulting in a beginning decline in the psychic life, a decline further hastened by a continuance of the disordered metabolism with its toxins diffusely attacking the nerve cells of the upper cortical layers, which are made more liable to destructive influences by the cutting off of their reflex sensory stimulation.

CEREBELLAR FITS

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This paper is based on the seizures that occurred in a series of forty-five cases of subtentorial brain tumor, in which the patients were operated on at the Neurological Institute during the past few years. Twenty-three were cases of cerebellar tumor, six were cases of tumor of the pons and midbrain, and sixteen were cases of the cerebello-pontile angle.

EXPERIMENTATION AND CEREBELLAR EXCITABILITY

Since the well-known experiments of Flourens with pigeons nearly a century ago, it has frequently been demonstrated that ablation of parts of the cerebellum may be followed by peculiar forced movements of the limbs and body. A dog with half of the cerebellum removed may circle about toward the side of the lesion. Monkeys with the inferior cerebellar peduncles divided, thrust their body and limbs about in an odd manner and assume various rigid and sustained attitudes.

The electrical excitability of the cerebellum is a matter for further investigation. Since the first experiments of Ferrier¹ many important studies have been made, notably by Pruss, Versiloff, Negro and Rosendo, Clark and Horsley,² Rothmann, Uffenorde and others. Some of the findings are uniform but many are contradictory. For instance, some observers (Pruss,³ Versiloff³) have obtained by electrical stimulation of the cerebellar cortex, certain motor responses of the neck, trunk and extremities, which in their general character resemble those obtained by stimulation of the cerebrum in that the contractions of isolated muscle groups were both tonic and clonic. Clark and Horsley, remarking that strong stimuli are required to evoke a decisive effect from the cortex of the cerebellum, as all experimenters have agreed, questioned whether in positive cases the results obtained might not be due to diffusion of the stimuli to underlying nuclei. Later these

1. Ferrier: Allbutt's System of Medicine 7:376, 1901.

2. Clark and Horsley: Brain 31:121, 1908.

3. Pruss, Versiloff, quoted by Tilney: Neurol. Bull. 2:303 (Aug.), 1909.

observers,² using a perfected technic with the bipolar method, concluded that the cerebellar cortex is not excitable.

As the problem of cerebellar excitability is still the subject of dispute, it seems to us impossible to derive therefrom any satisfactory conjectures as to the mechanism of cerebellar fits or the frequency with which they might be expected in lesions of the cerebellum, such as result from the growth of a tumor.

THE CEREBELLUM AND MOTILITY

André-Thomas⁴ calls attention to certain clinical and experimental observations which are of especial interest to the subject of this paper.

According to the observations of Horsley, the activity of the nerve centers is "translated" by a combination of clonus and tonus factors, and the resultant motor manifestations differ according to the proportions of one or the other of these factors. Clonicity is a property of the cerebral cortex and tonicity of the lower centers. The conclusions of Horsley and Bouché on this point were demonstrated experimentally. They injected essence of absinthe into the jugular vein of a cat three weeks after ablation of its left cerebral hemisphere. On the right side of the body, represented cortically in the ablated hemisphere, an attack of tonic contractions occurred, with the limbs in extension. On the left side, represented in the intact right cerebral hemisphere, a tonic-clonic attack of contractions was produced, with the limbs in flexion. During the course of an attack, the authors made an instantaneous section through the midbrain: immediately the clonic movements were changed into tonic movements of the whole body, the head was drawn backward and the limbs of the formerly flexed left side went into extension. Excitations of the inferior centers, among which is counted the cerebellum, gave rise to exclusively tonic attacks.

Some clinical facts sustain these observations. Hughlings Jackson⁵ cited the case of a child afflicted with a tumor of the median lobe of the cerebellum, in which tetanoid convulsive attacks were observed. The forearms were flexed on the arms, the arms were held close to the sides, the head drawn backward, the body was in a position of opisthotonos with extended legs. Jackson concluded from this: 1. In convulsions of cerebellar origin the spasm is tonic, whereas in cerebral convulsions it is principally clonic. 2. Cerebellar convulsions affect more the bilateral muscles of the legs and trunk, whereas in cerebral affections one side is more involved than the other and the arm more than the leg. 3. Cerebellar crises resemble tetanus more than epilepsy.

4. André-Thomas: *Cerebellar Functions*, New York, 1912, p. 167.

5. Jackson, Hughlings: *Med. Times & Hosp. Gaz.* 1:626, 1865.

This contrast between the clonic character of cerebral convulsions and the tonic character of cerebellar ones is to be compared, according to André-Thomas, with the special form that movement takes in persons with cerebellar atrophy. It becomes "discontinuous and clonic," apparently on account of the disappearance of the tonic cerebellar influence.

MAJOR CEREBELLAR SEIZURES

For the classic picture of what may be considered a major cerebellar seizure, we are indebted to the observations made years ago by Hughlings Jackson.⁵ He declared that attacks of tonic rigidity of muscles of the back of the neck, with retraction of the head, associated with flexion of the forearms and extension of the legs and pointing of the toes, were an especial feature of tumors of the middle lobe of the cerebellum and a direct result of cerebellar irritation.

Rigidity of the muscles of the neck and jaws, with rigid extension of the extremities and flexion of the arms, was noted by McCewen⁶ in cases of subtentorial abscess. Retraction of the head and rigidity of the masseters were reported in similar disease states by Drummond,⁷ Friedeberg⁸ and others long ago. While such symptoms are not unusual in inflammatory conditions in the posterior fossa, they have seldom been observed to occur with tumors of this region.

In Stewart and Holmes'⁹ series of forty cases of cerebellar tumor, there was one case, a large tumor of the under surface of the vermis, in which extensive hemorrhage occurred into the substance of the left cerebellar hemisphere during operation; whereupon, while the patient was still under the anesthetic, a rigid spasm of the whole body occurred which "was purely tonic in all the limbs and uniformly maintained for about three minutes." There was retraction of the head and opisthotonos. The face and respiration were undisturbed. Attacks similar to the first recurred frequently until death nine hours later.

Dana¹⁰ has described attacks that occurred in cases of tumor of the cerebellopontile angle, which consisted of loud tinnitus or a roaring sound, vertigo with a tendency to pitch or fall to the ground, sometimes sudden blindness or clouding of consciousness and, in severe attacks, tonic spasms of the extensors lasting from two to five minutes.

In our series, one case comes under the heading of major cerebellar seizures.

6. McCewen: *Pyogenic Diseases*, 1893, p. 197, Case 1.

7. Drummond: *Lancet* **2**:190, 1894.

8. Friedeberg: *Berl. klin. Wchnschr.* Aug., 1895, p. 719.

9. Stewart and Holmes: *Symptomatology of Cerebellar Tumors; A Study of Forty Cases*, *Brain* **27**:520, 1904.

10. Dana: *The Cerebellar Seizure (Cerebellar Fits), A Syndrome Characteristic of Cerebellar Tumors*, *New York M. J.* **81**:270, 1905.

CASE 1.—A. P., a girl, 11 years old, at the age of 3 began to have attacks of nausea and vomiting. A short time later some gait disturbance became noticeable: she did not lift her feet clear of slight obstructions. Occasionally she complained of pain over the mastoid region. The interesting feature is the occasional occurrence, after the first year of her illness, of attacks of general rigidity with opisthotonos, both arms flexed and raised, lower limbs extended, toes pointed. These attacks were accompanied by vomiting and marked nystagmus. It is remarkable that only at these times was nystagmus observed.

At operation (Dr. Elsberg) a cystic tumor was found involving the middle and right lobes of the cerebellum.

ATTACKS OF TONIC SPASM AND FORCED MOVEMENT

Stewart and Holmes reported a few seizures in large tumors of the pons and cerebellum, which were unlike anything they had seen in forebrain lesions in that the spasms were attacks of purely tonic rigidity quite different from the typical tonic-clonic spasms which result from instability or irritation of the cerebral motor areas. They also described attacks of jerking of an irregular shock-like character, in the homolateral arm in a case of one sided cerebellar tumor. According to the statement of the patient, the contralateral arm was also occasionally simultaneously affected but always in slighter degree.

None of our cases presented an example of general forced movements of the trunk, and it is likely that the forced movements which result from experimental ablation have nothing but an incomplete counterpart when the cerebellum is injured by a lesion such as tumor. Dr. Dana said that in one of his cases, that of a woman with a tumor in the posterior fossa, who, as a comparatively early symptom, would, in what he regarded as a forced movement, extend her body suddenly and slide from her chair to the floor.

Among our forty-five cases there were four which presented at some time or other, homolateral rigidity of an extremity with irregular spasmodic jerking movements.

CASE 2.—C., a girl, 15 years old, for six months had had increasingly severe attacks of headache, usually in the morning, occasionally accompanied by vomiting. The pain was most marked in the temporal regions, especially the right. During the past two or three months vision had been failing and during the past week dizzy spells had frequently occurred. Lately she had had occasional convulsive attacks of the left arm. The movements, unlike a jacksonian fit, were aimless, somewhat sudden stiff jerks, of an irregular or arrhythmic character, with no local point of commencement, no gradual spread to other parts and no clouding of consciousness. Operation (Dr. Elsberg) disclosed a tumor involving the left lobe of the cerebellum.

CASE 3.—L., a woman, 43 years of age, had noticed for ten years "a heavy tired feeling" in the right side of the tongue. For five years hearing in the right ear had been poor and she was troubled by a noise in the head "like

steam escaping from a radiator." At times she talked only with difficulty, as one intoxicated. For two years she had had peculiar attacks involving the right side, arm, and leg, which were quite unlike a jacksonian fit. The movements had no local point of commencement, no gradual spread, and were not rhythmic or clonic in character. The attack might be described as a sudden loss of control of the limbs on the right side, which trembled and were stiffly thrust about. A tumor of the right cerebellopontile angle was found at operation (Dr. Taylor).

CASE 4.—S. P., a woman, 31 years of age, for eighteen months had had increasing difficulty in walking, was ataxic and dizzy. For twelve months the muscles on the left side of the face had been weak. During the past month there had been severe attacks of headache with vomiting, mostly in the morning. During the past week the patient had been confined to bed.

Two attacks had occurred during the past week. The attacks were accompanied by intense headache and numbness of the left side of the face, and the patient was unable to speak although conscious. The movements had no local point of commencement, no gradual spread and were not clonic, but were sudden, jerky, spastic, of wide range, of irregular character, arrhythmic and confined to the left arm. At operation (Dr. Elsberg) a tumor was found in the left side of the cerebellum.

CASE 5.—M., a woman, 48 years of age, for years had had occasional severe general headaches. For the past few months she had had frequent occipital headaches and a more or less persistent pain in the back of the neck. For eight months her gait had become increasingly ataxic. She had marked tinnitus and dizzy attacks so severe as to cause her to fall. Her left leg had felt heavy at times during the past few weeks. Lately attacks involving the left arm had occurred which were not true convulsive movements but might be described as sudden seizures of ataxia or spastic unsteadiness. The fingers trembled and were unable to hold anything, and there was an associated unusual nodding or ataxic tremor of the head. There was no loss of consciousness. At operation (Dr. Elsberg) a tumor was found in the left cerebellopontile angle.

VARIOUS CRANIAL NERVE ATTACKS

Irritation of the cranial nerves about the pons and medulla by tumors in the posterior fossa, may cause striking symptoms of abrupt onset. Starr,¹¹ Sörgo¹² and others have described vagal attacks due to pressure on, or stretching of, the vagus nerve at the foramen magnum. Oppenheim¹³ has mentioned a case in which the patient had persistent contraction of the soft palate and vocal cords. Cushing¹⁴ speaks of "cerebellar crises" occurring in two of his thirty cases of cerebellopontile angle tumor. He describes them as paroxysms of extreme and agonizing type, with retraction of the neck and head, res-

11. Starr: *Am. J. Med. Sc.* **39**:552, 1910.

12. Sörgo: *Monatschr. f. Ohrenh.* **35**:285, 1901.

13. Oppenheim: *Text-book of Nervous Diseases*, Trans. by Bruce, Foulis, London, Ed. 5 **2**:907, 1911.

14. Cushing: *Tumors of the Nervus Acusticus*, Philadelphia, W. B. Saunders Company, p. 170, 1917.

piration difficulties, altered pulse, a sense of impending death and possibly unconsciousness. There are five other patients in his series who also had some "discomfort" of this kind, but in a much milder form.

Two of our patients had speech and swallowing difficulties, and one of them had also periodic choking sensations with respiratory embarrassment, but there were no patients in our series with severe "cerebellar crises," such as Cushing describes.

CASE 6.—K., a woman, 27 years of age, for four years had been troubled by a roaring sound in the right ear, which she compared to the sound of falling water. Since it began she had been almost deaf in this ear. Fifteen months before, while pregnant, dimness of vision had commenced; headaches with attacks of vomiting commenced about the same time. These symptoms persisted after the baby was born, and then in addition there came times when she would see double. During the past few months the patient had staggered toward the right. Her eyesight had continued to fail rapidly, and for the past few weeks she had been quite blind in the left eye and nearly so in the right. Lately she had had spells in which it was difficult for her to speak and sometimes difficult to swallow, accompanied by a panicky feeling as though she were about to choke. At operation (Dr. Elsberg) a glioma was found involving the right cerebellar lobe.

CASE 7.—Kr., a woman, 47 years of age, for two and a half years had had constant discomfort in the left eye: "It feels as though an eyelash were in it." At about the time that this came on, she also began to have a queer sensation in the left side of her face, "like a bug crawling." Until six weeks before there had been no headache, but since that time she had had considerable pain in the occiput and back of the neck. For one month she had been dizzy and ataxic, staggering toward the left. Her tongue had felt numb, there had been a constant sweet taste in her mouth and a discomfort in her throat which caused her to swallow constantly in an effort to get rid of it. There was an occasional roaring or whistling sound in her ears, especially in the left. Lately she had had difficulty in speaking and in swallowing, and a convulsive tic had commenced in the left side of her face, which in the past few days had become almost constant. At operation (Dr. Elsberg) a tumor was found in the left cerebellopontile angle.

FACIAL SPASM

Spasms of the face, which may be wrongly diagnosed as jacksonian epilepsy, may result from irritation of the facial nerve by tumors in the posterior fossa. Operations have been performed over the motor area on this account. Hughlings Jackson,⁵ in 1865, noted homolateral attacks of facial spasm which he had difficulty in accounting for. Stewart and Holmes mention them as possible though inconspicuous symptoms. Two cases are cited by Mills and Weisenberg,¹⁶ and some

16. Mills, C. K. and Weisenberg, T. H.: Cerebellar Symptoms and Cerebellar Localization, J.A.M.A. **63**:1813 (Nov. 21) 1914.

slight facial twitching was present in three of Cushing's patients, in one of them bilaterally.

In our series there are three cases in which an attack of homolateral facial twitching occurred. In none was it an early symptom, and it was otherwise unlike a jacksonian attack in that it had no local point of commencement with gradual spread. It remained limited to the face. On careful consideration the spasms in these cases will be found to conform more in character to those of facial convulsive tic (facial spasm) than to those of an epileptiform attack. It is likely, too, that some associated symptoms, such as involvement of other cranial nerves, will assist in making their subtentorial origin apparent.

CASE 8.—Mc., a woman, 45 years of age, for two years had suffered with headache and vomiting. She also had had intermittent pain over the eyes and left side of the face and tinnitus with comparative deafness of the left ear of gradual development. At times during the past year she had seen dazzling lights before her eyes and had been ataxic in gait. Two months before she had had severe twitching of the left facial muscles. At operation (Dr. Taylor) a tumor was found involving the left side of the cerebellum.

CASE 9.—W., a woman, 43 years old, three years before had commenced to feel weak, dizzy and nauseated at times. A year and a half before she had begun to have tinnitus, bilateral, but especially marked in the right ear. Her hearing had become poor in this ear, and she had had neuralgic pains in the right eye and right side of the face and head. During the past year she had had much headache, generalized and severe, and her vision had rapidly failed. In the past few months the right side of her face had felt numb, and her neck large and stiff. On one occasion, three months before, the right side of her face had twitched spasmodically for five minutes, preceded by an attack of dizziness. At operation (Dr. Elsberg) a tumor of the right side of the cerebellum was found.

GENERALIZED CONVULSIONS

In not one of our forty-five cases did generalized convulsions occur, nor were any reported in Stewart and Holmes' series of forty cerebellar tumors. This should be interesting to those who have considered an epileptic convulsion to be a common general sign of intracranial tumor, attributable, like headache, vomiting and optic neuritis, to heightened intracranial pressure, for it is a well-known fact that the intracranial pressure increases early in subtentorial tumors and usually to a marked degree.

We accept the view that a generalized epileptic convulsion in the last analysis is always the expression of one physiologic state (no matter what influences entered into the production of that state)—a more or less generalized cortical instability. This is in accord with the belief expressed years ago by Ferrier,¹⁷ one of the most experienced investi-

17. Ferrier: *Allbutt's System of Medicine* 7:375, 1901.

gators of cerebellar excitability, that if a generalized convulsion occurs in connection with a cerebellar lesion, it has an indirect relationship only, and is not due to direct irritation of the cerebellum. This is also the conclusion of James Collier¹⁸ in his important paper on "The False Localizing Signs of Intracranial Tumor." He tells of "several" cases of tumor of the brain stem and cerebellum, in which general convulsions occurred, " . . . but in all these cases the symptoms were of long standing when the convulsions first occurred, and in all of them the autopsies revealed considerable ventricular distention." He considers that the convulsions were the result of internal hydrocephalus which by pressure had produced an unstable condition of the cerebral cortex. Generalized convulsions as the result of cerebellar tumor are rare, and Collier's explanation for their occurrence may be accepted.

JACKSONIAN CONVULSIONS

No jacksonian attacks occurred in any of our forty-five cases, nor were there any spoken of among the forty cases of Stewart and Holmes. Collier,¹⁸ however, calls attention to two cases in which tumor of the cerebellum caused jacksonian attacks. Local convulsions of slow spread and confined to the arm and face were observed. No lesion except cerebellar tumor was present, but the ventricles were considerably distended, and some bilateral spasticity was present. The cases were of long standing when the convulsions occurred. Collier attributes the spasms to cortical instability, the result of pressure from internal hydrocephalus. He believes that the occurrence of such attacks should not lead to confusion in localizing the tumor, if signs of intracranial pressure have long been present, for cerebellar signs will probably have been conclusive early in the illness.

CONCLUSIONS

There are nine cases only in this series of forty-five posterior fossa tumors in which phenomena occurred that might be considered under the caption of "fits." From a study of these cases and a brief review of the literature, we have concluded that convulsive phenomena of any sort in tumors of the posterior fossa are rare; that the chief distinguishing feature of those which have been noted is the irregularity and sustained tonicities of the movements, in comparison with the rhythmic, clonic movements of forebrain fits.

Sudden involuntary movements, similar in a measure to the so-called forced movements which follow experimental ablation of parts of the cerebellum, were observed in some cases.

18. Collier, James: The False Localizing Signs of Intracranial Tumor. Brain, Pt. 14:490, 1904.

Sudden characteristic disturbances in the function of the cranial nerves in the posterior fossa, such as tic-like spasms and respiratory embarrassment, may result from irritation by a tumor in this region.

Fits of any kind usually occur late in the illness, after conclusive signs of cerebellar disease have long been present.

Jacksonian convulsions, which may result from instability induced by the growth of a tumor in the vicinity of the cerebral motor cortex, are easily distinguishable from cerebellar fits by the deliberate, progressive, clonic character of the spasms.

We wish to express our indebtedness to Dr. Elsberg, Surgeon to the Institute, for the privilege of studying the operating room records.

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LUMINAL THERAPY IN THE CONTROL OF EPILEPTIC SEIZURES

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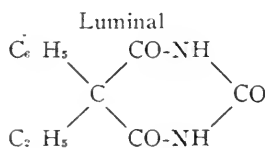
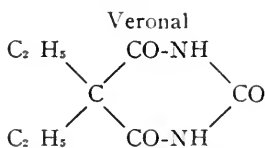
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Notwithstanding the many ingenious hypotheses advanced to explain epilepsy, it still must be assumed that it is a disease of unexplained etiology. In the present state of information, it might be best regarded as the result of an increased irritability of the cerebral cortex, occurring as an expression of organ inferiority, with the seizures as responses of this cortex to stimuli of exogenous or endogenous origin. The fact that most patients at first have nocturnal fits, then nocturnal and diurnal ones, and that there is a tendency toward an increase in the number of seizures, might best be explained on the principle that every nervous activity is enhanced by each preceding one; in other words, each epileptic seizure predisposes to the following one. While it takes considerable time for the highly irritable cortex to respond to the stimuli, when once it has responded, it is much easier for the following fit to come through; in other words, the threshold is lowered. It is therefore logical to utilize those agents that diminish the irritability of the cortex and to employ factors that minimize all stimuli. To accomplish this end is the goal of practically all who have to deal with the epileptic patient.

The drugs most frequently employed have been, in one form or another, preparations of bromid. It is true that in certain cases this drug has done good. In the vast majority of cases, however, it has failed and its after-effects have been most distressing. The picture of bromidosis is too well known to need description.

The drug that has given us most unusual results, and which we employed in the present study, is luminal. It is a phenyl-ethylbarbituric acid and is therefore closely allied to veronal.



Hauptman¹ was the first one to use this drug in epilepsy; he reported a series of cases in 1912. The dosage employed was 0.1 gm.

1. Hauptman, Alfred: Luminal bei Epilepsie. München. med. Wchnschr. 59: 1907 (Aug. 27) 1912.

in the morning and 0.2 gm. in the evening. In the milder cases, the seizures disappeared and in the severe cases they became milder in their manifestations. No postepileptic stupor followed the use of this drug, and there were no other undesirable after-effects. Many of the patients were able to return to their homes in good condition and to become self supporting.

Kutzinski² also studied a series of cases of patients with epilepsy to whom he daily gave luminal in doses of 0.1 gm. to 0.3 gm. He also noted complete disappearance or reduction in the number of the seizures, according to the severity of the case. He found luminal less efficacious in infantile epilepsy; in none of the cases was there improvement in the mental symptoms; and, as the effect of luminal was transitory, the seizures recurred on withdrawal of the drug. Nevertheless, there were no undesirable after-effects, such as reflex or pupillary disorders, cardiac or urinary disturbances.

Fuchs³ obtained satisfactory results in a series of thirty patients treated by luminal in doses of 0.075 gm., three times a day. The seizures ceased in most cases, but returned with greater intensity and frequency with the discontinuation of luminal. The best results with the drug were obtained in patients who showed a tendency to psychosis. The author concluded that luminal is the best anti-epileptic drug known at the present time.

Debrowski,⁴ choosing a series of epileptic psychoses for experimentation with luminal, found that favorable results were obtained, even in cases of long standing dementia. He gave doses of 0.1 to 0.2 gm., two or three times a day, occasionally giving 0.3 gm. twice a day. He believed there was no counter indication to its use, and no complication occurred, except that larger doses gave him unfavorable after-effects, such as drowsiness, slight ataxia and a slow scanning unintelligible speech, these symptoms clearing rapidly, however, on withdrawal of the drug.

Dercum⁵ found that luminal exercised a remarkable control over the seizures even in the most confirmed cases of epilepsy, usually inhibiting them promptly. He administered a grain and a half of

2. Kutzinski, A.: Luminalbehandlung bei Epilepsie, *Monatschr. f. Psychiat. u. Neurol.* **36**:174, 1914.

3. Fuchs, W.: Epilepsie u. Luminal, München. med. Wchnschr. **61**:873 (April) 1914.

4. Debrowski, W. G.: The Effect of Luminal in Epileptic Dementia, *Monatschr. f. Psychiat. u. Neurol.* **36**:248, 1914.

5. Dercum, Francis X.: On the Complete Control of Epileptic Seizures by Luminal, *Therap. Gaz.* **43**:609 (Sept. 15) 1919.

luminal, or two grains of sodium luminal at bed time, after putting the patient through a course of bromid preparatory treatment. There were no indications of any deleterious or untoward effects, and the results were most gratifying.

Our study of eighty-six cases of epilepsy covered a period of eight months, and it was limited to female psychotic epileptic patients.⁶ We did nothing to alter the general routine or the management of the patients; we merely administered sodium luminal in a definite dosage to all patients, each one receiving a sufficient amount of the drug to make an impression on the particular case. Previous to this, the patients received either bromid, hyoscin, chloral, or some other sedative to counteract the unpleasant effects associated with the seizures, such as assaultative tendencies, irritability, excitability, destructiveness, etc. We at first limited the use of the drug to fifteen of the most serious cases. These were selected either because of the unusual frequency of the seizure, or unusually distressing after-effects, such as great post-epileptic stupor, confusion, destructiveness and homicidal tendencies. Our results were so prompt and striking in this rather small group that after a month's trial we administered this drug to all patients in the epileptic group.

We used sodium luminal rather than the alkaloid itself because of its greater solubility. This has been of great advantage in our cases because many of the patients would not take medicine, and we therefore put the drug in their tea or coffee unknown to them. As a general rule, the dose employed was three fourths of a grain, three times a day, but each patient was carefully studied before a definite dosage was given. We found that while some patients required more than three doses a day, others did better on only one dose given just before retiring.

RESULTS OF ADMINISTRATION OF LUMINAL

The results obtained were striking. The first improvement noted was a decided decrease in the number of seizures. This could best be gaged when the total number of seizures in May, 1919, were compared with the number in May, 1920. There were 502 recorded seizures in May, 1919, while only eight seizures were recorded in May, 1920, in the same ward. While it is conceivable that there may have been a different and possibly a milder type of cases in 1920 than in 1919, the difference is entirely too great, even if we make such allowance. Furthermore, over 60 per cent. of the patients under our treatment were in the ward in May, 1919.

6. This study was conducted at the Manhattan State Hospital, Ward's Island, N. Y.

There was a definite diminution in the nature of the seizures, the convulsions being much milder and of shorter duration.

There was a definite decrease and change in the unpleasant after-effects of the seizures.

There was a definite favorable impression on the menstrual function of patients.

We were enabled to parole patients whom we otherwise would not have considered fit to leave the institution.

The patients became much quieter and more amenable to care and treatment.

A decided improvement in the general morale of the ward was observed. There was a decided decrease in the number of injuries received during seizures, and in the number of altercations with other patients. Less demands were made on the overworked nursing staff.

The after-effects of the drug have been practically nil. We had one case, that of a deteriorated young epileptic patient, who had as many as six convulsions in twenty-four hours. She was given luminal and was apparently doing well when we suddenly noted that she had become rather somnolent and did not respond to questions. There were no physical signs that could be attributed to the drug, such as ptosis or paralysis or sensory disturbances. She remained in that somnolent state for ten days, during which time she received no medication and had to be fed forcibly. At the end of this period the condition gradually cleared up, and she had an epileptic seizure. We then resumed the administration of the drug in diminished dosage, finally giving her three-fourths of a grain of sodium luminal every evening. She has continued to improve, has requested that she be allowed to do a little work in the ward and to go to church, and has brightened up remarkably.

There were no accumulative effects in any of the cases; there was no dulness, no exanthems, no paralysis, nothing that would lead one to believe that any of the toxic effects that have been reported by other observers were present.

This drug, however, cannot be considered free from all unpleasant results. With the discontinuation of its administration the seizures return at more frequent intervals and with greater intensity. This and the fact that as yet we are unable to explain the manner in which the drug acts would indicate that the drug should be used with caution and only by trained men. Furthermore, the margin of safety is rather small; German observers warn against the use of luminal in doses of more than 0.3 gm. in twenty-four hours. Toxic symptoms resembling those caused by veronal poisoning, drowsiness, slurring, scanning speech, paraphrasia, ataxia, dilated pupils, and gastro-intestinal symp-

toms—have been reported by Farnell⁷ and others. These observers, however, used the drug in too large a dosage and as an hypnotic to induce sleep. We believe there are much safer and more valuable drugs to be used for hypnotic purposes, because the dose required to induce sleep makes administration of luminal too dangerous. As an anticonvulsive, this drug at present is more nearly a specific one than any other we have at our disposal.

We did not neglect the generally accepted measures for diminishing the irritating stimuli sent to an easily excitable cortex; dental and eye defects were corrected and careful attention was given the bowels, the diet and bathing, as well as to the matter of air and exercise.

1198 Eastern Parkway, Brooklyn.

7. Farnell, F. J.: Luminal; Its Toxic Effects. *J. A. M. A.* **61**:192 (July 19) 1913.

News and Comment

COLORADO NEUROLOGICAL SOCIETY ORGANIZED

On December 20, the Colorado Neurological Society was organized with thirteen charter members. The officers are: president, Dr. Howell T. Pershing, Denver; vice president, Dr. Philip Work, Pueblo, and secretary and treasurer, Dr. George A. Moleen, Denver. Bimonthly meetings will be held.

Abstracts from Current Literature

ZUR KLINISCHEN PATHOLOGIE DES ZWISCHENHIRNS (CON-
TRIBUTION TO THE CLINICAL PATHOLOGY OF THE DIEN-
CEPHALON. ERICK LESCHKE, Deutsch. med. Wchnschr. **46**:959 (Aug. 26)
1920.

The author states that considerable controversy still exists concerning the function of the pituitary and of the diencephalon, due to their close relation. In the general discussion he recalls that Eckard pointed out that the destruction of the corpora mamillare resulted in polyuria, while in 1884 Loeb demonstrated that disease of the diencephalon caused disturbance in the heat-regulating mechanism and glycosuria. In 1891, Ott showed that by stimulation of the tuber cinerium heat regulation was altered, and in 1907 he further demonstrated that the strongest pyretic (such as tetrahydronaphthylamin) failed to produce fever after extirpation of the corpus striatum and tuber cinerium. Caselli found that by stimulation of the adjoining portion of the base of the diencephalon, following extirpation of the pituitary gland, he obtained the same results as Cyon obtained by direct stimulation of the pituitary gland, namely, slowing of the pulse, increased blood pressure, and in one case glycosuria. In this connection Winkler demonstrated that following the removal of an eye or section of the sympathetic fibers of the neck, a degeneration of certain cell groups and fiber tracts occurred in the hypothalamus.

Interest in the physiology of the diencephalon was first awakened by the work of Karplus and Kreidl in 1909. In their work, by electrical stimulation of the infundibulum, they obtained sympathetic reactions; dilatation of the pupils, widening of the palpebral fissure and retraction of the nictitating membrane. Edinger at this time also ascribed to the diencephalon sympathetic functions. Aschner, in 1912, in his exacting work on the physiology of the midbrain, was able to separate the functions of the pituitary gland from those of the diencephalon. He showed that following stimulation of the base of the diencephalon, glycosuria with polyuria, slowing of the pulse, contraction of the bladder, rectum and uterus and atrophy of the genitalia occurred. In 1916, he postulated from his work that a chemical and visceral center was present in the diencephalon, which, among other functions, had a special bearing on the clinical pathology of diabetes mellitus, insipidus, and dysplasia adiposo-genitalis.

In 1912, Krehl and Isenschmidt had also demonstrated that the heat-regulating mechanism was destroyed following section of the diencephalon, while in 1913 the author obtained similar results by midbrain puncture. Leschke further was able to demonstrate that the basal medial portion of the hypothalamus contained not only the normal heat-regulating power, but, in conjunction with Citron, demonstrated that the temperature center also belonged here, for following its separation neither infection nor pyretics would cause a rise in temperature. Later, in conjunction with Schneider, the author showed that by stimulation of the diencephalon, an arrest of protein metabolism occurred. This observation corresponds with the results obtained by Graffe. Karplus and Kreidl found that the diencephalon also exerted some influence over hidrosis. Müller and Glaser assume a vasomotor center to be in the diencephalon, which

when stimulated causes increased blood pressure and constriction of vessels.

The author next reviews the work on the basal ganglions of the midbrain, referring to the work of Wilson on lenticular degeneration, Levy on paralysis agitans in relation to the caudate nucleus and ansa lentiformis, and the more recent work of Frank in relation to the tonic innervation of striated muscle which depends on the sympathetic innervation.

1. In the discussion of the anatomy of the diencephalon the author refers to the work of Edinger in which this author shows that the diencephalon, especially the basal portions, belongs phylogenetically to the older portions of the brain stem. The diencephalon is found in the lowest vertebrates, forming in them the highest center of the nervous system, just as the cortex functionates in the highest vertebrates. Edinger considers the diencephalon a part of the primary mechanism of the brain, while the telencephalon phylogenetically is of much later development. The diencephalon ganglions develop from the central gray matter which is present about the middle ventricle in lower animals (third ventricle in higher vertebrates). The author states that the regio subthalamica stands in close relation to the cortex in the phylogenetic scale. In the lower vertebrates it is anatomically and functionally the highest brain part, controls all regulatory function, and acts in the lower vertebrates as the cortex does in the higher vertebrates. Evidence to support this analogy is seen in progressive paralysis in which both are equally degenerated.

2. Diencephalon and Diabetes Insipidus: According to the work of Aschner, diabetes insipidus is not the result of pituitary disturbance, but due to function of the diencephalon. Camus, Roussy and the author have noted polyuria following destruction of the base of the midbrain. In spite of the recent work in diabetes insipidus in which so much evidence has been brought to bear on the pituitary origin, among others the experiment in which extract of this gland produces in the healthy a polyuria, the author feels that all that may be ascribed to the pituitary may also be ascribed to the diencephalon. He gives these facts to show that the pituitary alone is not the cause of diabetes insipidus: (a) Extirpation of the pituitary in animals without injury to the diencephalon does not cause a constant or even transient polyuria. Following extirpation of the posterior lobe, one does not obtain the cachexia which follows extirpation of the anterior lobe. (b) Destruction or atrophy of all or part of the pituitary in man is not followed by diabetes insipidus. Following destruction or atrophy of the anterior lobe of the pituitary gland, a characteristic cachexia develops; also loss of weight, prematurity, apathy, even coma, secondary anemia and sexual impotence. Isolated destruction of the posterior lobe is not followed by any change, the same being true for the infundibulum. The author concludes that diabetes insipidus has no relation to pituitary function.

Concerning the relation between the diencephalon, especially the basal portion, and diabetes insipidus, the author gives these facts: (a) Puncture of the tuber cinereum directly posterior to the infundibulum will produce polyuria and, in the cases of pituitary disease showing midbrain disturbances, also diabetes insipidus. There are numerous verified cases of diencephalon disturbances in which the pituitary gland was not involved and in which diabetes insipidus occurred. Such cases have been noted following (1) gunshot wounds, (2) basal tumors, (3) softening in the midbrain, (4) gumma of the midbrain, (5) tubercles of the infundibulum, (6) pineal tumors, (7) vascular lesions, and (8) internal hydrocephalus.

The occurrence of polyuria during migraine or epileptic attacks indicates a cerebral influence in diuresis. The frequent occurrence of polyuria following

basal skull fracture is explained on the ground that as a rule the diencephalon is the portion that suffers. All these factors indicate that the basal (infundibular) portion of the diencephalon plays an important part in the pathogenesis of diabetes insipidus.

3. *Diencephalon and Diabetes Mellitus*: Against the pituitary theory of diabetes mellitus are the same points as in polyuria. (a) Extirpation of the pituitary gland or one of its lobes, if the diencephalon is not affected, does not cause diabetes mellitus in animals. (b) Change in the pituitary gland by tumor or atrophy in man does not bring about diabetes mellitus.

On the other hand, one finds that in disease of the base of the diencephalon and surrounding portions glycosuria is produced which may lead to polyuria, and the two conditions not infrequently occur simultaneously. The author found that in 42 per cent. of a group of 189 cases of acromegaly, a lasting alimentary glycosuria developed. He gives cases of basal ganglion lesions, apoplexy, basal skull fracture and basal syphilitic meningitis, in which polyuria with diabetes was present. To bear out this work, the author quotes from Borchard, Falta, Noorden and others.

As to the relation of the pancreas to diabetes, the author states that even if the pancreas is affected there is no reason to believe that the diencephalon does not also play a part. He states that in two such cases of diabetes mellitus in which disturbance in the pancreas was found, he also found changes in the diencephalon. The changes in the brain were confined chiefly to the hypothalamus. The author feels that changes in the diencephalon can be expected only in a portion of the cases of diabetes mellitus, for the diencephalon represents only one of the stations of the sympathetic system. The possibility of a sympathetic nervous system connection of the midbrain and the pancreas and liver is brought up by the author.

4. *Midbrain and Dystrophia Adiposia Genitalis*: Since Fröhlich in 1901 presented the syndrome bearing his name and its relation to pituitary function, it has withstood all attacks. As early as 1904, Erdheim opposed the pituitary theory in Fröhlich's syndrome. He pointed out that in several cases the pituitary gland showed neither macroscopic nor microscopic changes. He believed that adiposity resulted from a functional disturbance in a trophic center situated in the diencephalon in the region of the infundibulum. Aschner was of the same opinion, and showed that by destruction of the base of the diencephalon without injury to the pituitary gland an atrophy of the genitalia took place.

Against the pituitary theory of dystrophia adiposia genitalis, the author makes these statements: 1. In thirty-five cases of Fröhlich's disease that came to necropsy, there were at least twelve in which the pituitary gland was not involved; this is also true of two additional cases of Winkler and four of Oberndorfer. 2. In the remaining twenty-three cases a most varied form of tumors in the pituitary structure led to the syndrome. 3. Loss of pituitary function in man, when limited to the anterior lobe, is followed by characteristic cachexia (Simonds); disturbance of the posterior lobe is followed by no change.

Concerning disturbance of the diencephalon in cases of adiposia genitalis the author says: 1. In all cases of dystrophia the diencephalon is affected even if the pituitary is not involved. 2. Following isolated destruction of the diencephalon in animals, genital atrophy occurred; adiposity was not noted. 3. Destruction of the midbrain caused increased protein metabolism. Stimulation of the diencephalon, on the other hand, caused decreased protein metabolism. 4. Dystrophia adiposia genitalis is at times found in conjunction with other disturbances of the diencephalon, such as polyuria and low temperature.

The author feels that these facts point to the presence of a metabolic center in the diencephalon. The relation between the pituitary gland and the diencephalon in this disease must remain an open question.

5. *Temperature Disturbances in Diseases of the Diencephalon*: Loeb, as early as 1873, and again in 1884, called attention to the tuber cinerium in temperature disturbances. The author states that if one reviews the literature concerning pituitary tumors with or without acromegalia, one finds many cases in which there is an unusually low temperature, and in some cases, unexplained periods of high temperature. This was noted in some of Cushing's cases; he attributed the temperature change to the pituitary gland. Leschke points out, however, that in animals in which the pituitary gland is removed, or in man, in cases of atrophy of the pituitary, the heat regulating mechanism is left intact. The author states that he has frequently seen cases of pituitary tumor with rise in temperature, in which the temperature could not be explained either clinically or pathologically. Diseases of the tuber cinerium may make impossible a high temperature. As an illustration the author cites a case in which a patient with pneumonia at the onset had a temperature of 39 C., and on the following days of only 36.8. At necropsy examination it was found that the diencephalon was infiltrated with round cells and that there was an area of softening in the tuber cinerium. Max Meyer found in a case of localized encephalitis of the corpora mamillaria, besides polyuria, a temperature of 38 C. The high temperature following apoplectic seizures localized in the diencephalon and fractures of the skull with polyuria and glycosuria tend to show that the diencephalon has something to do with the heat regulation.

6. *Diencephalon and Eye*: After the removal of an eye or section of the cervical sympathetic or superior cervical ganglion, there occurs a degeneration of the falciform cell groups of the hypothalamus below the floor of the third ventricle, also of the fibers in the central gray matter under the nuclei habenulae, while stimulation of the midbrain produces dilation of the pupils and separation of the lids. These findings have not been noted in pituitary tumors, and the author believes the reason for this may be that the early involvement of the optic nerve at the chiasma overshadows the above findings.

Among other interesting cases the author quotes one of Oppenheim's, who observed in an adenocarcinoma of the pituitary gland which came to necropsy, blindness of the right eye with partial blindness in the left. The interesting feature was the fact that in the blind eye reaction of pupil remained, while in the partially blind eye the pupil reflex was lost. Oppenheim did not attempt to explain the finding, and the author thinks that the midbrain was compressed and thus led to a central disturbance.

7. *Relation of the Diencephalon to Vascular Innervation and to Hidrosis*: The author reviews the work of several authors, among others, that of Karplus and Kreidl, who showed that stimulation of the base of the diencephalon produced hidrosis. This has not been observed clinically. The hidrosis is supposed to be due to a stimulation of sympathetic fibers in the diencephalon.

The same observers were able to show that stimulation of the diencephalon caused a contraction of the blood vessels. Following the withdrawal of the stimulation, the blood vessels showed marked congestion. While these authors do not state that there is a vasometer center in the diencephalon, the author feels that his own observations in this regard are conclusive. Following the injection of tetrahydronaphthylamin he obtained contraction of all peripheral vessels and high temperature, up to 45 C., while following section of the diencephalon neither of these reactions were obtained. There is little clinical evidence of

vasomotor action of the diencephalon, but the author calls attention to Aschner's work in which he raises the question whether there is any relation between migraine and the diencephalon. The author has observed a case of injury to the base of the skull in which diabetes insipidus, transient glycosuria, vasomotor paralysis and unilateral hidrosis were present. The occurrence of migraine with polyuria, vasomotor disturbances and other disturbances of the diencephalon are also frequently noted clinically.

The author concludes that even though the clinical pathology of the diencephalon rests on a hypothetical base, and the relation of the diencephalon and pituitary gland have not been fully worked out, one cannot doubt, in the face of the evidence that has been brought forth, that alterations in the function of the diencephalon, especially of the hypothalamus, may lead to important changes in the vegetative economy, showing themselves in the form of diabetes insipidus, diabetes mellitus, dystrophia adiposogenitalis, heat regulatory changes, disturbances of pupillary reaction, hidrosis and vasomotor activity.

MOERSCH, Rochester, Minn.

REPORT OF A CASE OF EXTRADURAL AND SUBDURAL ABSCESS FOLLOWING SUPPURATING FRONTAL SINUSITIS AND OSTEO-MYELITIS OF THE FRONTAL BONE. JOSEPH H. BRYAN, Am. J. Med. Sc. **160**:5 (Nov.) 1920.

This is a case report of a boy, aged 15, who gave a history of "nasal catarrh" during the preceding winter and at the first examination, Jan. 13, 1919, showed a swelling over the left frontal sinus and pus appearing beneath the middle turbinate of the left nasal cavity. His temperature was 100 to 101. The sinus was opened and a large amount of pus evacuated; then it was curetted and drained. He did well for several days, though the pus drained profusely. Two weeks later he developed an abscess of the left upper eyelid and, because of the large amount of drainage, was reoperated. A sequestrum of bone was removed from the frontal region leaving an opening into the cranium with a small point of exposure of the dura. A third operation became necessary and was performed April 15. A fruitless search was made for diseased bone to account for the profuse and continued drainage. At this time he also developed a right maxillary sinusitis. On the third day after the second operation the cavity was flushed out with boric acid solution when it was being dressed; later, headache, numbness in the right hand and thickened speech developed. These conditions, however, cleared up. On May 6, the whole left frontal region was swollen, edematous and bloody with purulent secretion from the fistulous opening. There was no headache, the temperature was normal, and the boy said he felt well. A fourth operation was performed the day following the examination, and pus was found beneath the periosteum: the bone beneath it was necrotic up to the frontal eminence. The diseased bone was removed and the cavity packed. Secretion lessened slowly, although the wound was apparently healing well. Convalescence was slow. Late in July, while dressing the wound, a probe was passed to detect diseased bone, but instead it went through the perforation into the cranium and, on being withdrawn, the secretions flowed more freely. The wound was kept open and draining during August, and the boy's general condition remained excellent, with no headaches or other symptoms. In September the wound was closed except for the fistulous opening at the inner angle of the orbit. A canula

inserted along this tract and through the perforation of the skull passed up to the vertex, following which 6 drams of pus mixed with blood were evacuated. This was done every other day with copious evacuations and the roentgen-ray, with the canula in situ, showed that the abscess was either in the frontal lobe or between the lobe and skull. Another roentgenogram taken after a 10 per cent. solution of nitrate of thorium had been injected showed a large extradural abscess. On September 25 another operation was performed, and the abscess contents were evacuated, the walls gently curetted, painted with iodine and a drainage tube inserted. Nonhemolytic streptococci were found on bacteriologic examination. Progress was favorable, and the patient was discharged in ten days. Secretion gradually subsided and by December had apparently ceased. On Jan. 5, 1920, he was reported as very ill with influenza, bronchitis and inflammation of the accessory sinuses. He recovered from this and by February 14 was in good condition, except for pallor of the skin. On February 16 he was severely ill; he had a septic appearance; his temperature was 103 F.; there was a swelling over the left frontal region, and he had severe headache. The next day he had nystagmus of the right eye, twitching of the muscles of the face, nausea and a leukocytosis of 23,000. An operation was performed that evening which revealed that the old abscess sac was filled with pus and closed off. Some necrotic bone was found and removed. The patient reacted well from the anesthetic, but remained semicomatose. There was slight drainage. On February 17 the spinal fluid showed 370 cells, but no organisms. There was some loss of motion of the right arm. Two days later there was twitching of the right facial muscles, nystagmus in the right eye, involuntary evacuations, 16,000 leukocytes, and the spinal fluid contained 49 cells, but no organisms. The twitchings then extended to the right arm and leg and convulsions set in, continuing at intervals until his death on the afternoon of the 23d. Neurologic notes made the day before death showed weakness of the entire right side of the body, a positive Kernig's sign, inability to name objects and misplacing of words. The convulsive seizures were of the focal type always beginning on the right side of the face.

Necropsy showed a thickened left dura and the left hemisphere covered with pus, with a center at the junction of the Rolandic and Sylvian fissures, at which point the brain substance was depressed. Bacteriologically this pus contained streptococci. The anterior half of the left hemisphere showed a depression of 1 cm., the pia much thickened generally and marked vascular injection.

PATTEN, Philadelphia.

PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN UEBER DIE ENCEPHALITIS LETHARGICA, MIT BESONDERER BERUECKSICHTIGUNG IHRER STELLUNG ZUR GRIPPE-ENCEPHALITIS (PATHOLOGIC-ANATOMIC INVESTIGATIONS IN LETHARGIC ENCEPHALITIS, WITH SPECIAL REFERENCE TO ITS RELATION TO INFLUENZAL ENCEPHALITIS). RUDOLF JAFFÉ, *Med. Klin.* 16:1013 (Sept. 26) 1920.

In this present article Jaffé reports forty cases. The pathologic findings in lethargic encephalitis are so well known that he does not wish to discuss them, but he wonders whether lethargic encephalitis is to be considered a specific disease entity, or whether it is possible that it is an accompanying picture of influenza.

In the author's material, males and females were about equally affected. The youngest patient was 16, two patients were over 50; most of the patients were between the ages of 30 and 50.

While agreeing in the main with those of Economo, there are points of difference, especially in the more unusual findings. The macroscopic picture is usually negative. Occasionally one finds more or less hyperemia of the membranes and brain substance, especially of the gray matter. The only other macroscopic findings are hemorrhages.

Microscopically as a constant finding the author mentions a perivascular infiltration of the smaller and larger vessels, especially in the brain stem, and most marked along the aqueduct, floor of the fourth ventricle and medulla. The remainder of the brain and the spinal cord are frequently free from change. Jaffé has not seen a single patient with lesions in other portions of the brain, in whom the portions mentioned above were not affected, and always to a more severe degree. If other portions of the brain are involved, the order as found by him is: central ganglions, spinal cord, and last the cerebral cortex; the cerebellum in his cases never showed any pathologic condition.

The infiltration is confined to the adventitia. It does not invade the brain substance. The cells are chiefly round cells with sparse protoplasm and large nuclei, rich in chromatin, in type resembling lymphocytes. The vessels are usually filled with blood, contain no white cells, and do not show changes in the intima with cell infiltration. Because of this the author feels that the infiltration is not blood borne, but that the cells are adventitial lymphocytes in the sense of Marchand. Plasma cells are rather rare. Only occasionally does the infiltration invade the adjacent brain substance.

The extent of the infiltration along a blood vessel is hard to determine without serial sections. Economo has seen cases in which the infiltration about a blood vessel in the cortex has sharply disappeared as the vessel entered the white substance. Jaffé has never noted this. He has seen an infiltration branch off and follow certain vessels while neighboring vessels were unaffected. Arteries may be affected as well as veins, the latter more frequently, however. In typical cases this infiltration is always present. Other pathologic conditions are seen but never without the vascular changes described.

The second important, and not at all rare finding, consists in the presence of hemorrhages. It is frequently found associated with the vascular infiltration, and usually about the vessels infiltrated. At times hemorrhages are noted without any indication of blood vessel change. An infiltration of the brain substance may occur more or less localized, which has also been described by Economo and Oberndorfer. The two above forms are not regularly found, being present in eleven of Jaffé's cases. In both forms altered glia cells, lymphocytes and occasional plasma cells, also infrequent polymorphonuclear leukocytes are found. These isolated infiltrations can be differentiated by the absence of a central vessel and should a vessel be contiguous it will be normal. Occasionally such an infiltration will abut a perivascular infiltration and one can distinctly see the typical round cell perivascular infiltration with the infiltration showing plasma cells and polymorphonuclear leukocytes.

The final main finding is neuronophagia. On this finding Economo lays great stress and because of it differentiates lethargic encephalitis from all other inflammatory processes of the central nervous system, with the exception of poliomyelitis. Oberndorfer does not think that neuronophagia is especially typical of lethargic encephalitis but may also be seen in other processes. Jaffé

is not certain that this is one of the important findings in the picture of this disease.

The meninges are seldom involved. The infiltration consists in round cells about the blood vessels, but the pial tissue may be invaded and hemorrhages may occur. The author states that it has frequently occurred that in relatively young people great numbers of corpora amylacea were found.

The author states that he attempted to differentiate the lethargic encephalitis cases from influenzal encephalitis cases, but it was practically impossible to determine to which group the individual case belonged, as he saw cases that came to necropsy diagnosed as lethargic encephalitis which showed a typical hemorrhagic encephalitis, while influenzal encephalitis cases showed distinct findings of lethargic encephalitis. Most of his patients gave a history of influenza. Five had influenzal pneumonia. In only three cases was encephalitis the only finding. Thus he could divide his cases into only two groups: patients with hemorrhages and patients without hemorrhages. As Economo had already mentioned the occurrence of hemorrhages, the close relationship between lethargic encephalitis and hemorrhagic encephalitis became evident. Jaffé asks the question, "Do cases of hemorrhagic encephalitis following grippe show pathologic changes similar to those found in lethargic encephalitis?" This he answers in the affirmative, for he states that there are cases in which a distinct combination of the findings of hemorrhagic and lethargic encephalitis occur. Thus he finds that several cases have come to necropsy with the clinical diagnosis of encephalitis and meningitis following influenza in which were seen macroscopically hemorrhages into the white substance of the brain, but in which microscopic examination showed typical pictures of lethargic encephalitis. The possibilities of the two conditions coexisting is brought up by the author, but the great frequency and the grades of admixture seem sufficient to the author to put the two diseases on the same basis. He feels that they are two different forms of the same disease, and that any combination of the two pictures is possible.

A rather constant finding in lethargic encephalitis is the vascular infiltration which usually occurs in the gray substance. Why this should occur is not known. The author also reports findings that are rather at variance with those previously reported. He has seen cases running a typical lethargic encephalitis course in which there was none or little infiltration, but that in numerous blood vessels many polymorphonuclear leukocytes and leukocytic thrombi were found.

Jaffé does not believe that one can consider lethargic encephalitis as a disease picture *sui generis*. Clinically the symptoms are variable. The lethargic is only the predominating symptom in a portion of the cases: frequently, it is absent entirely. Motor symptoms, twitchings, chorea, myoclonia, etc., may be present. In two cases of chorea gravidarum, findings typical of lethargic encephalitis were obtained. Homen has recently investigated a series of non-purulent infectious toxic cases of encephalitis in which there were no findings not present in lethargic encephalitis. The author believes that one can come to only one conclusion, namely, that other toxic encephalitides may produce the same picture as lethargic encephalitis and that lethargic encephalitis is also of toxic infectious origin. Jaffé does not believe that Economo's experiment with monkeys is conclusive. In this experiment Economo obtained a hemorrhagic encephalitis by the intradural injection of pleomorphic streptococci. In spite of this the author does not feel that from the pathologic-anatomic

standpoint lethargic encephalitis is to be considered as an isolated picture. He sees in it an infectious toxic encephalitis which may occur after any infectious disease. In conclusion, the author states that while lethargic encephalitis presents a fairly typical picture, it cannot be distinguished from the hemorrhagic encephalitis of influenza or other forms of toxic infectious encephalitis, and that the general term infectious encephalitis might better be used, appending the name of the exciting disease, whether pneumonia, typhoid or influenza.

MOERSCH, Rochester, Minn.

TWELVE CASES OF THROMBOSIS OF THE CAVERNOUS SINUS.

J. JULIAN CHISHOLM and S. SHELTON WATKINS, Arch. Surg. **1**:483 (Nov.) 1920.

From a study of 50,000 surgical histories, the authors report twelve cases of cavernous sinus thrombosis, giving a detailed account of the onset of symptoms, etiology, physical examination on admission including examination of the blood, urine, teeth and tonsils, course in the hospital, treatment, result and necropsy findings when necropsy was granted.

The anatomy of the venous supply of the head is reviewed, especially the relation of the cavernous sinus to the lateral, petrosal and longitudinal sinuses, the ophthalmic vein, pterygoid plexus and the facial vein. The authors also point out its relationship to the cranial nerves, sphenoidal sinus and pituitary body. A knowledge of this anatomy gives a clearer insight into the clinical symptoms which result from an obstruction of the cavernous sinus.

The three most common causes of thrombosis of the cavernous sinus are: (1) marasmus, (2) trauma, and (3) infection. The latter is by far the most usual, and was the cause in all twelve cases reported.

Septic thrombosis usually begins with phlebitis, after which the regular phenomena of thrombi develop, with a red, adherent friable mass occluding the lumen, which may become purulent. The walls become thickened. Usually both cavernous sinuses become involved, but one before the other. There may be a basilar meningitis, meningeal hemorrhage, brain abscess and emboli involving the lungs, spleen, kidneys and other parts of the body. Usually there is more or less infection of neighboring vessels and nerves.

Symptoms of cavernous thrombosis may be due to (1) venous obstruction, (2) involvement of neighboring nerves and (3) general sepsis. Those due to venous obstruction are exophthalmos, edema of the retina, eyelids and bridge of the nose. Both eyes may be affected, but usually one is affected before the other, the second within forty-eight hours after the first. Symptoms may almost entirely disappear in one eye, while developing in the other. Associated with retinal edema, there is usually tortuosity and dilatation of the veins, retinal hemorrhages, and at times a low grade choked disk. Clouding of the media and opacity of the cornea occur shortly after the onset of eye symptoms. Edema of the lid may be so great as to close the eye completely.

When the pterygoid plexus is affected, edema of the pharynx and tonsil on the same side results; and edema of the skin over the mastoid and neck occurs when the lateral sinus and jugular vein become thrombosed, and edema of the face, when the facial vein is involved.

Cranial nerve symptoms are: ptosis, restricted ocular movements, dilatation of the pupils, loss of vision, and pain in the head due to the ophthalmic division of the trigeminal nerve. Ptosis and restricted ocular movements are due to paralysis of the third, fourth and sixth cranial nerves, while pupillary

symptoms may be due to paralysis of oculomotor or stimulation of sympathetic.

The septic symptoms are: temperature ranging usually from 101 to 106 F., rapid, small and thready pulse, chills and sweats. Vomiting, delirium and coma may also be present. Other symptoms are those of meningitis, pulmonary embolism, infection of the kidneys, liver and spleen.

Sometimes the symptoms of thrombosis are masked by those of the focal infection, especially when it begins in the orbit, when there is edema, exophthalmos, etc.

Diagnosis is based on a history of chills, headache, septic temperature and an exophthalmos, that always begins on one side, but usually involves the second eye within from twenty-four to forty-eight hours.

Erysipelas, cellulitis of the orbit secondary to nasal sinus infection, and tumors of the orbit may at times simulate septic thrombosis of the cavernous sinus, when only one eye is involved.

Treatment consists in the removal as soon as possible of the focus of infection. There is a wide difference of opinion about operating on the cavernous sinus itself, as only 7 per cent. of the patients recover without operation and less than 7 per cent. with operation. The principal methods of operation are: (1) by the temporal route, (2) by the orbital route, (3) by the ethmoidal-sphenoidal route, and (4) through the antrum of the opposite side.

The authors thus summarize their findings:

1. Operation was not attempted in any of the cases reported.
2. If operation is to be attempted the choice of route to be employed should depend on the focus of infection, whether in the orbit, ethmoid or sphenoidal cells, etc.
3. Failure of surgical intervention is due to the fact that basilar meningitis and thrombosis of the neighboring venous sinuses usually occur early in the disease.
4. Lumbar puncture is important, as often there are meningeal symptoms with a clear fluid. If the fluid is clear there is some chance for the patient without an operation, whereas if examination of the spinal fluid reveals the presence of a septic meningitis, the mere draining of the cavernous sinus will be of no avail.

SCARLETT, Philadelphia.

A STUDY OF THE RELATION BETWEEN THE REPRODUCTIVE ORGANS AND DEMENTIA PRAECOX. T. MATSUMOTO, J. Ment. Sc. **66**:414 (Oct.) 1920.

Sir Frederick Mott presents for the author an unusually interesting histologic study of the testicles and seminal vesicles in 100 cases of mental disease. The psychoses were well distributed, including defective states, paresis, manic-depressive psychoses, senile dementia and dementia praecox. The findings in dementia praecox were important enough to be repeated in detail.

"Numerous specimens of testes were examined from twenty cases of this disease. They may be divided into three groups, roughly speaking, according to the time between onset of symptoms (as far as could be ascertained) and death. This examination led to the general conclusion that the earlier the symptoms came on and the longer their duration before death, the more pronounced were the histologic changes.

In the first stage of regressive atrophy only a few of the tubules show morbid changes, the most obvious being a diminution in size and fewer spermatogenic

cells, with fewer cells showing active nuclear mitosis, absence of spermatids and spermatozoa. The Sertoli cells are seen much more distinctly resting on the thickened basement membrane. The interstitial tissue in the region of the atrophied tubules, generally speaking, is correspondingly increased. The interstitial cells containing lipid granules can be seen and numbers of lipid granules are observable in the Sertoli cells.

In the second stage many more tubules are similarly affected, but there may still be some tubules showing all stages of spermatogenesis. Examined with an oil-immersion lens the heads of the newly formed spermatozoa, both in the first and second stages, show appearances suggestive of degeneration. They are often of irregular shape and staining reaction; they present appearances like the degenerated forms described by Sir Frederick Mott as occurring in the fluid from the vesiculae seminales of cases of dementia praecox. Often they have an oxychromatin instead of a basichromatin reaction with the hematoxylin and eosin dyes. In fact, there appears to be a general deficiency of the basichromatin reaction of the nuclei of the spermatogonia and spermatocytes in all the tubules in this second stage.

In the third stage, which constituted the greater number of the twenty cases examined, there is almost complete or quite complete arrest of spermatogenesis. In the most advanced cases (and they are especially those which were admitted to the asylum in very early adolescence) the tubules show a very thickened basement membrane and no spermatogenic cells; a few Sertoli cells are seen within the tubule and an empty sustentacular network. Stained with Scharlach R., numbers of large, coarse droplets of fatty matter of various sizes are seen in the spaces. The interstitial cells of Leydig can be seen in the first two stages containing fatty droplets, but they appear to be less numerous and less distinct in their outline than those seen amidst the atrophied tubules in the testes of general paralytics. In the third stage, the cells of Leydig are still more difficult to find and the interstitial lipid is less observable. The interstitial connective tissue in some of the cases has undergone proliferation, and it is not uncommon to find therefore a fairly large testis in which there is a complete regressive atrophy of the spermatogenic cells. In other cases there is no interstitial connective-tissue proliferation. In all the cases, however, there is thickening of the basement membrane, and instead of one layer of flattened nucleated cells there are several.

The microscopic picture in the other psychoses was much less significant and contrasted strongly with the schizophrenic findings. The cases of paresis and senile dementia are especially valuable as controls against the influence of advanced age and various terminal conditions. The inference is that there exists in dementia praecox a primary regressive testicular atrophy which is distinctive and does not occur in other psychotic states. This seems to be a strong conclusion, but is justified by the author's careful work. Two explanatory hypotheses are presented: First, there are biochemical changes in the reproductive organs and nervous system, probably of nuclear origin and dependent on a germinal inborn defect of nuclear durability. Second, a defect in the germ cells may lead to a disorder of the balance of the endocrine functions, resulting in a disturbance of the normal nutritional equilibrium of the neurons with hypofunction and decay. To the reviewer this seems the more plausible. In any event, there is a new argument in favor of the essentially organic nature of dementia praecox.

STRECKER, Philadelphia.

LA PARALYSIE GENERALE EST DUE A UN TREPONEME DISTINCT DE CELUI DE LA SYPHILIS BANALE (GENERAL PARESIS IS DUE TO A DISTINCT TREPONEMA). A. MARIE and C. LEVADITI, Rev. de méd. **37**:193 (April) 1920.

The authors recall that the likelihood of the development of general paresis, in a case of syphilis, is in inverse proportion to the occurrence of peripheral ectodermic reactions. Fournier has concluded that general paresis follows, in a habitual quasiconstant fashion, the syphilis of benign initial type. The authors have previously detailed many examples wherein the appearance of tabes and general paresis was found in subjects infected with syphilis from a known common source. Erb cites an instance in which five men, infected by the same prostitute, all became either paretic or tabetic. Nonne, Brosius, and Babinski have reported similar observations. From such observations, one can deduce, at least, the theory of a neurotropic form of syphilis with a special nerve tissue affinity.

In general paresis, there is the constant presence of *Spirochaeta pallida* in the cerebral cortex and its frequent existence, though probably intermittently and ephemerally, in the blood and spinal fluid. The authors have succeeded in causing three successive passages of the virus of general paresis in rabbits. They used the blood of a paretic patient for the initial inoculation. Results obtained with brain substance and with spinal fluid from paretic cases are also mentioned. With initial virus from a chancre they have obtained, in rabbits, the regular passage of infection over a period of six years. The following important differences between the neurotropic virus and the dermatropic, are discussed:

1. The inoculation period in the inoculation from man to rabbit ranged from forty to forty-five days with an average of forty-two with the dermatropic virus, while with the neurotropic virus it was ninety-five days as an average.
2. The incubation period in the inoculation from rabbit to rabbit averaged fifteen days with the dermatropic and seventy-five with the neurotropic virus.
3. The lesion produced with the dermatropic was an indurated chancre with microscopically intense infiltration, abundant connective new formation, a network of spirochetes at the base of the lesion and endo-arteritis and peri-arteritis. In contrast, in the neurotropic virus lesion, there was slight infiltration, no new formation of connective tissue, spirochetes in the epithelial layers, and only slight ulceration and desquamation of the epidermis.
4. The dermatropic virus, obtained originally from a chancre, inoculated into a rabbit, is transferable to monkeys. The neurotropic virus, obtained from the blood of a patient with general paresis, inoculated into a rabbit, is not then transferable to monkeys.
5. The dermatropic virus passed into a rabbit is transversible to man, as has been demonstrated by two accidental happenings. The neurotropic virus passed into a rabbit is not then transferable to man, as a voluntary attempt at inoculation showed.

Furthermore, the authors have found that rabbits inoculated with the dermatropic virus later became immune to that virus, but retain susceptibility to the neurotropic virus, and vice versa.

Judging by the results of these experiments and observations, the authors believe that the spirochete of general paresis must be considered as a different variety than the spirochete causing cutaneous and visceral syphilis.

DAVIS, New York.

REMARQUES SUR LE TRAVAIL DE M. LAFORA-TRAITEMENT INTRARACHIDIEN DES AFFECTIONS SYPHILITIKES ET PARASYPHILITIKES DU SYSTEM NERVEUX (REMARKS ON THE WORK OF LAFORA-INTRA-ARACHNOID TREATMENT OF SYPHILITIC AND PARASYPHILITIC CONDITIONS OF THE NERVOUS SYSTEM). G. MARINESCO, *Rev. Neurol.* **26**:901 (Dec.) 1919.

The author reports having obtained excellent results in the treatment of tabes and general paresis, particularly in early cases, by the subdural injection of autogenous serum arsphenamized *in vivo*.

Marinesco's technic may be outlined thus: From 75 to 90 cg. of neo-arsphenamin are administered to the patient intravenously, provided there is no history of seizures or evidence of cardiac or renal complications. After a period, varying from several hours to three days, 30 c.c. of blood are withdrawn and, without centrifugation, are set aside in the refrigerator overnight. The serum (from 10 to 15 c.c.) is then separated and, after inactivation, is injected subdurally, an equal quantity of spinal fluid having first been withdrawn.

Three protocols are submitted. In the first case, that of a woman, aged 33, with a history of syphilitic infection ten years previously, there was noted, prior to treatment, change of character, irritability and expansiveness, speech defect, tremor of the tongue, lips, and hands, writing defect, unequal pupils with loss of reaction to light, and hyperkinetic tendon reflexes. The spinal fluid showed positive Wassermann reaction, pleocytosis and increased solids. Six injections (10 c.c.) of auto-arsphenamized serum were administered at intervals of a week, and the course was repeated after three months. Following this, it was noted that the mental disturbance had apparently disappeared entirely, and the patient was able to resume her former occupation (photographic retouching). The spinal fluid Wassermann reaction was now but feebly positive, and there was no longer evidence of pleocytosis or increased solids. The pupillary changes, however, persisted. The patient, at the time of writing, had been under observation five years without return of symptoms.

In the second case, a man, aged 35, also with history of syphilitic infection ten years previously, there was observed marked depression with hypochondriacal features. The spinal fluid showed lymphocytosis, increased solids, and a positive Wassermann reaction. After two courses of six injections, as in the first case, there was noted apparently complete abatement of mental symptoms and marked diminution in the spinal fluid cell count (40 to 5) and solids content, although the Wassermann reaction showed no change. The patient was able to resume his occupation (physician). Four years later, while in army service, the patient suffered a relapse which terminated fatally. During the latter period he had apparently not been under the author's care.

In the third case, that of a man with syphilitic infection of long standing (age and specific duration not given), there was remarked, prior to treatment, elation of the expansive type, pupillary irregularity and tremor of the hands. The spinal fluid Wassermann reaction was positive, and there was marked lymphocytosis (270) and increased solids. After two courses of injections, as in the other two cases, the mental symptoms seemed to have greatly subsided, and the cell count was reduced to 12. The spinal fluid Wassermann reaction was now only weakly positive, and the increase in solids radically cut down. As this patient came under treatment only a short time prior to the preparation of this paper, no opportunity was afforded for observation as to the permanency of the improvement.

RAPHAEL, Kalamazoo, Mich.

ETUDE ANATOMO-PATHOLOGIQUE DES CENTRES NERVEUX DANS UNE CAS DE MYXOEDEME CONGENITAL AVEC CRETINISME (THE STUDY OF THE PATHOLOGIC ANATOMY OF THE CENTRAL NERVOUS SYSTEM IN A CASE OF CONGENITAL MYXEDEMA WITH CRETINISM). PIERRE MARIE, C. TRÉTIAKOFF and E. STUMFER, *L'Encephale* **15**:601 (Nov.) 1920.

The authors have undertaken a microscopic examination of the brain of a typical cretin in whom the thyroid was quite absent, being replaced by two cysts, each about the size of a hazelnut. The patient was well oriented, but was unable to read or write. She had been at the Salpêtrière six years. She was able to make her bed but was unable to do any work, such as knitting, sewing, etc. She died of an infectious diarrhea at the age of 36.

The authors were unable to make a thorough examination of the more delicate structures of the central nervous system, since the tissues had been in formaldehyd for over a year. The lesions which they were able to show consisted of a marked infiltration of the walls of the blood vessels of all calibers by an amorphous substance colored dark-violet by hermatein. This substance was either a compound of iron or calcium. By means of the reaction of Perls, the authors demonstrated that the coloration was due mainly to iron compounds.

Changes were found in the cerebellum, dentate nucleus and the lenticular nuclei. Examinations of sections of various other parts of the cerebral hemispheres, the oval center of Vieussens, pons, cerebral peduncles, medulla and spinal cord showed no changes. The authors suggest that this may have been due to the age of the specimen and the consequent technical limitations.

The walls of the blood vessels of these structures (cerebellum, dentate nucleus and lenticular nuclei) were infiltrated by the dark staining granules mentioned in the foregoing. The granules in some cases were as large as red blood cells. In the globus pallidus, the knee of the internal capsule, and the inner third of the putamen the lesions were most conspicuous. No area of softening was found.

In conclusion, the authors ask whether the accumulation of iron may not be related to the hypothyroidism. They emphasize the important part that iron plays in oxidation. They also mention the chromatolysis found throughout the nervous system by Mott and Bruns, and correlate this with the work of Marinesco, who showed that the chromatophil bodies contain much iron. They feel that the question is a difficult one and necessitates a solution by the biochemists.

The authors feel that their observations throw light on the origin of the cerebellar symptoms described in myxedema by Odier. They believe that the marked variations in the degree of mental impairment in many cases may be explained by the intenseness and localization of the vascular lesions.

KRAUS, New York.

INFANTILE SPINAL PROGRESSIVE MUSCULAR ATROPHY (WERDNIG-HOFFMANN). REPORT OF A CASE WITH NECROPSY FINDINGS. E. J. HUENEKENS and E. T. BELL, *Am. J. Dis. Child.* **20**:496 (Dec.) 1920.

The literature on infantile progressive muscular atrophy is reviewed and the clinical and pathologic pictures of the Werdnig-Hoffmann type are contrasted with the amyotonia congenita of Oppenheim. The conclusions formed,

in general, are that the two diseases are extreme grades of one and the same disease, having the same type of pathologic alterations in muscle and spinal cord, but varying clinically in some respects and with many gradations.

The following case is reported: There was myopathic disease on the mother's side in three first cousins, but the paternal history was negative. The first child, aged 8, was healthy; the second, died at 3 months of age, having given evidence of myotonia at birth; the case of the third child is reported; the fourth, aged 1, was normal.

The patient was born at full term, and appeared normal until the age of 6 weeks when its movements began to grow weaker, especially at the hips; the arms were later involved, and the whole condition progressed gradually. At 4 months, breathing and nursing were difficult. The authors saw the case for the first time when the child was 5 months old. He was underdeveloped and undernourished, with a flabby musculature, pale skin, and difficult breathing; the chest was deformed; the knee jerks were absent, and he had frequent spells of collapse. A mediastinal growth was suspected; later it was found that he had an enormously enlarged thymus. The child died at the age of 6 months.

NECROPSY REPORT: Atrophic muscles, enlarged thymus—extending to the diaphragm, distinctly lobulated, and weighing 29.5 gm.; lungs markedly collapsed; suprarenals greatly reduced in size and weight; other structures negative grossly.

MICROSCOPIC EXAMINATION: The spinal cord showed a few atrophic cells in the anterior horns in the cervical region, marked decrease of cells in the anterior horns in the dorsal and lumbar regions, and the few cells present were atrophic. There were no hemorrhages and no lymphatic infiltration. There was moderate reduction of the medullary sheaths in the fasciculus gracilis. The muscles showed distinct atrophy, with atrophic fibers intermingling with the normal. The thymus was hyperplastic and the lungs atelectatic.

The interesting features are that the case is familial and hereditary, and a typical Werdnig-Hoffmann type. The second child had a case of true amyotonia congenita of the Oppenheim type, showing that the two types existed in the same family, thus leading further to the establishment of the contention that the two diseases are merely extreme grades of the same disease.

PATTEN, Philadelphia.

ARTHRITES AIGUES PLASTIQUES ET MENINGITE CEREBRO-SPINALE A MENINGOCOQUE C (MENINGITIS, MENINGOCOCCUS TYPE C, WITH ACUTE PLASTIC ARTHRITIS). R. J. WEISENBACH and L. MERLE, *Progrès méd.* **35**:527 (Dec. 4) 1920.

The apparition of articular phenomena in the course of epidemic cerebro-spinal meningitis has been described by several writers, and the meningococcus has been isolated from affected joints by Fronz. The clinical aspect of the arthropathies have included an arthralgic form, a hydarthritic as well as a hemarthritic form and a suppurative form. The author reports a case in which the arthritic signs did not correspond to any one of the above. Instead there was clinically what the author calls a plastic arthritis, quite like an acute gonorrheal arthritis. The changes were entirely peri-articular, with local redness, heat and tenderness; there was no fluctuation.

The report concerns a woman of 23 ill with epidemic cerebrospinal meningitis, due to meningococcus type C, as shown by culture of the spinal fluid.

who forty-eight hours after the appearance of meningeal symptoms developed an arthritis of each wrist joint. Six days after the disappearance of meningococci from the spinal fluid, a third articular localization, of the left shoulder joint, developed, accompanied by a return of fever. The patient made a complete recovery by the end of fifteen weeks.

DAVIS, New York.

AMYOTONIA CONGENITA (OPPENHEIM). REPORT OF A CASE, WITH FULL HISTOPATHOLOGIC EXAMINATION. JAMES B. HOLMES, *Am. J. Dis. Child.* **20**:405 (Nov.) 1920.

The author reviews the literature on the subject with special reference to the underlying pathology. He concludes that "the significant lesion seems to lie in the cells of the anterior horns, in the large motor ganglion cells, and that most distinctly in those regions where these cells normally reach their greatest development, i. e., in the cervical and lumbar regions."

The case is reported fully and gives clinically a typical picture of amyotonia congenita as described by Oppenheim in 1900. The necropsy findings were the interesting features of the paper. The diaphragm was the only muscle appearing normal grossly and microscopically. In the nervous system the cord showed anterior roots diminished in size with a ratio of 1:3 and 1:4 with the posterior roots (normally 1:2), but otherwise normal configuration and size of the cord itself. Microscopically no evidence of recent degeneration or absence of myelination was found. The cells of Clark's columns were normal, but the anterior horn cells were few in number and smaller in size, although having a normal appearance in other respects. The glia cells showed no increase. The neurofibrillae in the anterior gray matter were somewhat diminished. The posterior roots were much better myelinated than the anterior, but no degenerative changes were seen. The peripheral nerves appeared normal. In the muscular system bundles containing small immature fibers, hypertrophied fibers and collections of cells resembling embryonic muscle structure, were found in the same muscles, but there was no evidence of degeneration or increase of adipose tissue.

The author comments on the absence of evidence of disintegration of muscle tissue, and considers the possibility of the presence of either a simple atrophy or, of "an example of retarded development." As the condition was present at birth (evidenced by impaired movement), all other factors in the production of atrophy can be ruled out except abnormal innervation. The striking pathologic feature—hypertrophied muscle fibers, and collections of small cells (embryonic), side by side in the same muscle shows a lack of uniformity of structure. In addition, the inability to demonstrate replacement tissue, strengthens the point of view that the whole is a defective developmental process.

PATTEN, Philadelphia.

LE HOQUET EPIDEMIQUE (EPIDEMIC HICCUGH). SICARD and PARAF; SUR QUELQUES CAS DE HOQUET PARAISSANT EPIDEMIQUE (CASES OF APPARENTLY EPIDEMIC HICCUGH), LOGRE and HEUYER; reported by MME. ATHANASSIO-BÉNISTY from the *Soc. de Neurol.*, meeting of Dec. 2, 1920; *Presse méd.* **28**:901 (Dec. 11) 1920.

Sicard and Paraf reported a series of twenty-two cases of hiccough collected over the preceding fortnight. Hiccough began suddenly with a minimum

of constitutional symptoms, continued almost without remission but with frequent exacerbations at all hours of the day or night, and finally ceased entirely without complications after two or three days.

Physical means of relieving or lessening the spasm were rhythmic traction on the tongue, pressure on the eyeball, mechanical distention of the esophagus, or an icebag over the course of the phrenic in the neck. Dufour showed several similar cases last year, at the time of Sicard's communication on myoclonic encephalitis. It was notable that this epidemic of hiccough was apparently paralleled by some recrudescence of epidemic encephalitis.

Logre and Heuyer called attention in their series to a nasopharyngeal catarrh, which seemed to precede the phrenic symptoms through a period of from twenty-four to forty-eight hours. The hiccough was intractable to therapy, interfered with eating but finally ceased during sleep, and lasted altogether about two days. The syndrome included some gastro-intestinal disturbances, fatigability, anxiety, and a slight febrile reaction. The rapid multiplication of similar cases suggested an epidemic infection, conceivably a benign phrenic type of influenza.

HUDDLESON, New York.

SCLERODERMA AS A POSSIBLE MANIFESTATION OF CHRONIC ARSENIC POISONING. SAMUEL AYRES, JR., *Arch. Dermat. & Syph.* 2:747 (Dec.) 1920.

Three cases of diffuse scleroderma were investigated, and in each instance contact with arsenic was established over varying periods of time although scleroderma began in one case before the exposure. Examination revealed arsenic in the urine of each patient. The typical lesion of scleroderma was determined in each case and reported fully. The duration of the disease is interesting in consideration of the possibility of arsenic acting as the etiologic factor; one year, four years and thirteen years, respectively. The etiology of scleroderma, so far as is known, is briefly discussed. The possibilities of arsenic are taken up, mention being made of lack of data concerning the frequency of finding of arsenic in the urine of people under ordinary conditions or suffering from some disease unrelated to arsenical poisoning. It is pointed out that urine will show arsenic from three to nine months after exposure and it is also observed that the chemical is intermittently excreted by the kidneys even in fatal cases. In the Massachusetts General Hospital arsenic was found in twelve out of twenty-five cases, 48 per cent., where arsenic was not suspected as a cause for symptoms, and in 43 per cent. of forty-eight specimens of urine collected at random.

The sources of arsenic poisoning are interesting. Arsin gas, which is highly toxic, is a frequent offender, being generated by the growth of certain species of molds on decaying wall paper, bread crumbs, etc. A similarity of symptoms common to generalized scleroderma and arsenical poisoning are: neuritic manifestations—numbness, tingling, soreness, etc.; pigmentation; changes in the skin itself; loss of weight; muscular weakness; intermittent, irregular fever; gastro-intestinal disturbances; vasomotor instability and rapid and irregular heart.

The author suggests that cases of scleroderma be studied from this point of view with the idea of establishing the exact etiology of the disease and also the investigation of arsenical poisoning as a "possibility."

PATTEN, Philadelphia.

SURGICAL EXPERIENCES WITH AN INTRACRANIAL APPROACH TO CHIASMAL LESIONS. GEORGE J. HEUER, *Arch. Surg.* **1**:368 (Sept.) 1920.

Heuer reports twenty-four cases in which an intracranial approach to the hypophysis through a large frontoparietal flap was used—an intradural approach in contradistinction to the original extradural method of Frazier. He believes that an intracranial approach best exposes, and can most adequately deal with, the greatest number of chiasmal lesions. He bases this opinion largely on the tendency of hypophyseal lesions to grow upward and to extend into the cranial chamber. He believes that the transsphenoidal approach should be considered only in those early cases in which there are sellar headaches but no neighborhood symptoms. Heuer frankly admits that the majority of surgeons are not of this opinion, that they believe the transsphenoidal approach is the best one because it is the simplest and because it results in the lowest mortality. So far, it has been impossible to remove all of the diseased tissue in dealing with hypophyseal lesions, and therefore any operative procedure that carries with it a primary mortality of 37 per cent. and a total mortality of 46 per cent. hardly seems to be justified in the hands of most surgeons. Heuer admits the mortality just mentioned; therefore we feel like leaving the future development of the operation to him, hoping that it can be made safer. In the meantime, most neurologic surgeons will continue to feel that saving the eyesight in dealing with hypophyseal lesions is of prime importance and will continue trying to do so by using the transsphenoidal method of approach.

RODMAN, Philadelphia.

PARALYSIES AMYOTROPHIQUES DISSOCIEES DU PLEXUS BRACHIAL A TYPE SUPERIEUR CONSECUTIVES A LA SERTHERAPIE ANTITETANIQUE (DISSOCIATIVE AMYOTROPHIC PARALYSIS OF THE BRACHIAL PLEXUS, SUPERIOR TYPE, FOLLOWING ANTITETANIC SERTHERAPY). J. LHERMITTE, *Rev. Neurol.* **26**:894 (Dec.) 1919.

Lhermitte reports three cases in which a subcutaneous (abdomen) injection of 10 c.c. of antitetanus serum was followed in from four to twelve days by dissociative amyotrophic brachial palsy, superior type. In one case, generalized urticarial eruption preceded the neurologic disturbance by twenty-four hours. The onset was abrupt in all three cases, and was accompanied by severe pain in the arm and shoulder in two of the cases. The paralysis, at first, included the entire arm but later, in each case, showed a tendency to limit itself to the muscular elements supplied by the fifth and sixth cervical roots with marked amyotrophic change. There was noted reaction of degeneration and marked diminution of osseotendinous reflex response but no sensory disturbance, except the initial pain, which was present in two cases and which persisted from two to six weeks. There was no evidence of myofibrillation or spasm.

The clinical course of the paralysis and the amyotrophic change was one of slowly progressive improvement requiring from six to nine months for even moderate restoration of function.

The only therapeutic procedures employed were massage and ascending doses of strychnin.

RAPHAEL, Kalamazoo, Mich.

BRAIN SWELLING. MARTIN REICHARDT, *Allg. Ztschr. f. Psychiat.* **75**:34 (March) 1919.

In an extensive "Referate" Reichardt, under several headings, presents a systematic discussion of the subject of brain swelling. In a general consideration, he discussed individual differences in the size of the cranial cavity and the evidence indicative of brain swelling. He defines brain swelling as an increase in the volume of the brain that is not the result of hyperemia or of increased free fluid (brain edema, hydrops meningens, hydrocephalus), and not the result of histologic changes in the form of a tumor or tumor-like growth, or of the usual inflammatory processes of suppuration or abscess formation. In discussing the histologic changes in the brain accompanying swelling, he emphasizes that the physical chemical change of swelling is something essentially different from brain swelling due to the increase in volume from hypertrophied or swollen elements, such as the presence of the ameboid type of glia cells described by Alzheimer. There are cases of brain swelling in which no histologic change is found to explain its occurrence. In discussing the causes of brain swelling, he states that the condition may occur in most of the exogenous and endogenous diseases of the brain. It is a reaction in a healthy brain as a result of some exogenous injury, but sometimes, as in epilepsy and catatonia, without any external cause that can be recognized. In these cases it seems to be related to the brain disorder that is present. There is a fundamental difference between brain swelling due to exogenous and to endogenous factors. The remainder of the first section of his review is devoted to the consideration of the clinical manifestations and the localization of brain swelling. The second section of the review deals with brain swelling in its relation to brain injury and traumatic brain diseases. In the third and fourth section are considered the constitutional factors involved in the process and the technic of the examination of the brain from its physical aspect. In the final section he makes a plea for a more general use of physical methods of investigation of the brain. His conclusions are that what one at present means by the term brain swelling is probably only a general concept. It is a special form of disease or reaction of the brain, a process that belongs in the general pathology of the brain, a group of anatomic changes that may be brought about by different diseases and causes of disease.

BARRETT, Ann Arbor, Mich.

DYSGENESIE PYRAMIDO-CEREBELLEUSE FAMILIALE (FAMILIAL PYRAMIDO-CEREBELLAR DYSGENESIA). D. PAULIAN, *Rev. neurol.* **26**:815 (Nov.) 1919.

The author reports two cases, occurring in a brother, aged 26, and a sister, aged 31, with gradual onset in both at 20. The picture was marked by the initial development of tingling and numbness of the extremities, particularly of the legs, followed by staggering, wide-based, ataxic gait, and, ultimately, practical loss of locomotion and disturbance of sphincter control. On examination, there was determined, in both cases, asthenia, practical inability to walk, bilateral Babinski reflex and ankle clonus, hyperkinetic tendon reflexes, particularly of the lower extremities, adiadochocinesis, dysmetria, intention tremor, bradyphasia and retention. Blood and spinal Wassermann reactions were negative, as well as cytologic examination of the spinal fluid. In addition, in the brother, there was sexual impotence, and, in the sister, cessation of menses.

The mother was syphilitic and a paternal uncle is reported to have suffered many years with what is described, somewhat indefinitely, as a "painful affection of the spinal cord."

Paulian directs attention to the resemblance borne by his cases to the clinical picture presented in hereditary cerebellar ataxia, but, in view of the absence of definite history of heredity, chooses to designate the condition exemplified by his cases as familial pyramidocerebellar dysgenesis, emphasizing, at the same time, the possible etiologic importance of parental syphilis in the production of the disturbance.

RAPHAEL, Kalamazoo, Mich.

ISCHEMIC NECROSIS OF THE HEART MUSCLE IN AN EPILEPTIC PATIENT DYING DURING AN ATTACK. GRUBER and LANG, *Arch. f. Psychiat.* **61**:99, 1919.

The authors report the case of a soldier who following an epileptic attack had been placed for observation in a hospital. Death occurred a few days later, six hours after a convulsive attack in which there was extreme irregularity of the heart. Necropsy revealed a comparatively recent ischemic necrosis of the heart muscle without changes in the coronary arteries or elsewhere in the heart. The opinion is advanced that the necrosis resulted from a spasm of the coronary arteries that probably had some relation to the epilepsy. The authors comment on the fact that not infrequently aurae and strange sensations referable to vessel spasm occur. Some observers have spoken of a "vasomotor aura," and have regarded the precordial sensations occurring in epilepsy as a direct expression of vasomotor constriction. Attention is directed to the occurrence of angiospastic conditions in neuropathic persons, such as Raynaud's disease and cases of gangrene of the skin, not infrequently seen during the war. Similar vascular spasms seem to be the cause of some cases of gastric and duodenal ulcers. The cause of the angiospasm in these conditions is central or reflex. In epilepsy it is not unlikely that the excessive nervous discharges may involve the central vasomotor apparatus.

BARRETT, Ann Arbor, Mich.

L'ABOLITION DU SIGNE DE BABINSKI PAR LE FROID ET SA REAPPARITION PAR LA CHALEUR (ABOLITION OF THE BABINSKI SIGN BY COLD AND ITS REAPPEARANCE AFTER HEAT). NOICA and A. RADOVICI, *Rev. Neurol.* **26**:891 (Dec.) 1919.

The authors report a positive Babinski reaction in a hemiplegic patient following the application of heat (hot-water bottle and immersion in heated water) to the foot although, on initial examination, the patient's feet having been somewhat chilled and cyanosed at the time, the response was consistently negative. Following the application of cold (ethyl chlorid), it was found that a positive response was no longer obtainable.

In two other cases, with uniformly positive Babinski reaction, the first that of a hemiplegic patient and the second that of a paraplegic patient, in which there had been determined, in addition, complete anesthesia extending from the soles to the level of the nipples, Noica and Radovici were able definitely to abolish the Babinski sign, for the time being, through the application of cold (ethyl chlorid) to the plantar surfaces. In the discussion of this last case, in which positive Babinski response was associated with predetermined anesthetic change, attention is called to the possibility of dissociation between conscious and reflex sensibility.

No mention is made as to whether or not the so-styled normal plantar flexion response was determined in the first case, prior to the application of heat and, in all three, subsequent to the application of cold.

RAPHAEL, Kalamazoo, Mich.

INVOLVEMENT OF THE NERVOUS SYSTEM DURING THE PRIMARY STAGE OF SYPHILIS. UDO J. WILE and CLYDE K. HASLEY, J. A. M. A. **76**:8 (Jan. 1) 1921.

The results of the examination of the spinal fluid during the primary stage of syphilis should be of considerable interest to neurologists. Of 221 fluids, forty-nine gave slight but unmistakable evidence of involvement of the central nervous system. In eight there was a positive Wassermann reaction; in twelve a pleocytosis and in twenty-five increased albumin and globulin. Since the fluid changes promptly disappeared under intravenous therapy, the authors infer merely a transitory meningeal roseola. The ease with which the early nerve involvement clears up under treatment, and the stubbornness with which it resists in late syphilis, again places strong emphasis on the necessity not only of thorough but of prompt treatment.

STRECKER, Philadelphia.

EXISTE-IL DES TROUBLES IRRITATIFS EN PATHOLOGIE NERVEUSE ET MENTALE? (ARE THERE IRRITATIVE DISTURBANCES IN NERVOUS AND MENTAL PATHOLOGY?) D. TRIANTAPHYLLOS, Rev. Neurol. **26**:881 (Dec.) 1919.

The author concludes that there does not exist a specific neuropathologic lesion type corresponding to the so-called "irritative lesion," which has been held by some to manifest itself in augmented neuropsychiatric function, as opposed to the "destructive" type, which tends to abolish function in the part or area involved. All lesions, Triantaphyllos contends, are essentially destructive in nature and tend, primarily, to bring about abolition of function but, on the other hand, they may secondarily give rise to "irritative" phenomena through the diminution or loss of the inhibitive control normally exercised over outlying centers by the areas actually involved in the destructive lesions. In other words, irritation, in the author's conception, represents not a primary "irritative" augmentation of function but rather a secondary disturbance of structurally uninvolved areas due to release from normal inhibitive control.

RAPHAEL, Kalamazoo, Mich.

UEBER DIE ERGEBNISSE DER PSYCHIATRESCHEN UND NEUROLOGISCHEN UNTERSUCHUNGEN AUF EINER KRANKENSAMMELSTELLE (THE RESULTS OF PSYCHIATRIC AND NEUROLOGIC EXAMINATIONS AT A HOSPITAL CENTER). HANS BERGER, Monatschr. f. Psychiat. u. Neurol. **47**:335 (June) 1920.

The report deals with observations made in 1915 at a hospital center close to the front which received its patients directly from combatant units. In the nine months covered by the report, there were 12,218 admissions; of these, 971 had mental or nervous disorders. Among the groupings are: dementia praecox, 17; depressive states (including melancholia, exogenic depressions, etc.), 34; manic depressive states, 8; paranoia, 2; severe constitutional psychopathic states, 2; chronic alcoholism, 29; genuine epilepsy, 48—34 of these

patients had had previous attacks, whereas 14 developed the first attack in the field. In the latter group, there was no hereditary factor noted. Probable genuine epilepsy was found in 47; traumatic epilepsy, 30; neurasthenia, 179 (the majority being constitutional neurasthenics); nervous exhaustion, 45; neurasthenic symptoms of undetermined origin, 65; mixed organic cases (multiple sclerosis, myelitis, etc.), 55; hysterias (developing without sudden fright), 56; conditions attributed by the patient to shock caused by an exploding bomb, hand grenade, etc., 233. Of these, 130 showed hysterical manifestations and 103 neurasthenic symptoms.

Of the 971 cases, functional nervous disorders comprised 592, or 60 per cent. The number of cases of acute onset, due to sudden fright were 233, or 24 per cent.

The author brings out an interesting relationship between the time of onset of the functional cases and the epilepsies in relation to active combat. The functional cases develop immediately; cases of epilepsy, after an interval. During a period of active fighting, the peak of admissions for epilepsy came one week after the peak of admissions for hysteria.

SELLING, Portland, Ore.

THE EFFECT OF MERCURY SALICYLATE ON THE WASSERMANN REACTION: OBSERVATION ON THE SEROLOGY OF EIGHTY-SEVEN PREVIOUSLY UNTREATED MEN. HERMAN GOODMAN, Arch. Dermat. & Syph. **2**:193 (Aug.) 1920.

This is a group study of the effect of mercury salicylate on eighty-seven patients who had never received treatment, and all of whom gave a positive Wassermann reaction. The patients received 1 grain of the drug at weekly intervals for a period of from six to eight weeks, and the second Wassermann test was made at the end of the first four weeks of rest. The results were: 47 patients showed no change; the reaction in 5 advanced from +++ to +++++; the reaction of 7 was reduced from +++++ to +++; the reaction of 1 was reduced from +++ to ++, of 5 from +++++ to ++, of 4 from +++++ to doubtful; the reaction of 9 patients became negative; and the reaction in 4 cases in which it had been negative became +++++. In all there was a change in 29 cases; there was no change in the remaining cases.

The author concludes that the effects produced were not remarkable, and that further treatment and an increase in dosage is indicated in all cases.

PATTEN, Philadelphia.

DE L'ALTERATION DU LIQUIDE CEPHALO-RACHIDIEN DANS LES PARALYSIES DIPHTHERIQUES DU VOILE DU PALAIS ET A TYPE DE POLYNEURITE (CHANGES IN THE SPINAL FLUID IN DIPHTHERITIC POLYNEURITIS AND IN DIPHTHERITIC PARALYSIS OF THE PALATE). LAVERGNE, Bull. méd. **34**:983 (Nov. 6) 1920.

Lavergne has found hyperglycorachia and albuminocytologic dissociation in cases of diphtheritic paralyses, whether general or localized.

The dissociation may be complete, with increase of albumin in the absence of cells, or partial, with a slight but disproportionate cellular reaction. These spinal fluid alterations appear early but last throughout the duration of the symptoms. They indicate that in the diphtheritic paralyses there is, as a rule, a meningeal reaction.

DAVIS, New York.

THE MENTAL HYGIENE OF THE INDUSTRIAL WORKER. CARL SCHEFFEL, *J. Indust. Hygiene* 2:5 (Sept.) 1920.

The general attitude of the workingman is that he must get as much as possible for his work—the largest financial reward in the shortest possible time. With his concept that labor is merely labor, he does not appreciate the relationship from an economical and social point of view, to society, and accordingly maintains a distinctly unhealthy attitude. Conversely, the employer has a duty toward the laborer that is not sometimes grasped. He should aim to keep labor clean and to keep the laborer busy and amused. The interest created by the employer in the workingman for the work that is done is reciprocal in results.

Labor, or work, is divided into skilled, unskilled, monotonous and diversified. Skilled labor carries with it a certain amount of mental satisfaction (or reward), namely, the finished product, creditably done. Work that shows no results carries no mental satisfaction. Most unskilled labor is monotonous; there is no mental stimulation in it and the work becomes mechanical; and the worker becomes introspective—to the detriment of his own morale and the results accomplished. Discontent and an unhealthy mental attitude is bred by lack of interest.

The author questioned thirty-one workmen in the plant where he is employed as physician, and by their replies to the question "What are you thinking about at this moment?" found that only three were thinking about the work they were doing.

All workers present three cardinal needs: adequate and systematic physical exercise daily, mental recreation of the right sort, and a good night's sleep. Having all these things, discontent is largely eliminated. Promotion is also a stimulus to interest and a basic factor in mental satisfaction. Employers should not make promises to their employees which cannot be kept. Their deeds are sufficient promise in themselves and engender interest and ambition.

PATTEN, Philadelphia.

CONTRIBUTION A L'ETUDE DES DELIRES TOXI-INFECTIEUSES: L'ONIRISME HALLUCINATOIRE; SES RAPPORTS AVEC LA CONFUSION MENTALE (CONTRIBUTION TO THE STUDY OF TOXICO-INFECTIOUS DELIRIUM; HALLUCINATORY ONEIRISM; ITS RELATIONSHIP TO MENTAL CONFUSION). R. CHARPENTIER, *Rev. neurol.* 26:755 (Oct.) 1919.

Charpentier believes that, contrary to the common conception, although frequently found together in toxico-infectious conditions, oneirism and mental confusion, aside from this common etiologic basis, have no essential relationship and may occur equally well singly. Psychopathologically the two processes may be distinguished in that in the so-called primary mental confusion attention is generally much impaired while in hallucinatory oneirism it is, if anything, definitely exalted. Amnesia is typical of the former and characteristically absent in the latter. In mental confusion, the disturbance is fundamentally of the more essentially intellectual mechanisms while in oneirism it is expressed chiefly in the sensorial field. One must be careful, the author cautions, not to confound the true primary mental confusion, occurring in toxico-infectious conditions with what he terms the secondary confusion frequently conditioned by the oneirismic disturbance. This should not be difficult

if one bears in mind the episodic character of the latter, its lesser duration as compared to the duration of the delirium, its later appearance and early disappearance, and, finally, its general mildness, with no disorientation.

RAPHAEL, Kalamazoo, Mich.

LEFT HANDEDNESS IN EPILEPTIC, FEEBLEMINDED AND NORMAL PERSONS. RUDOLF GANTER, *Allg. Ztschr. f. Psychiat.* **75**:589 (Aug. 14) 1919.

The author has studied this question in a series of 146 epileptic patients, equally divided between the two sexes, and 155 feeble-minded patients. Among the epileptic patients, 21.9 per cent. were left-handed and 18.7 per cent. among the feeble-minded. Studies of the families of patients of these groups who, while not themselves left-handed, had members of their families who were, showed that left-handedness was present in 45.9 per cent. of the families of epileptic patients and in 45.8 per cent. of those of feeble-minded patients.

In a series of ninety-three normal families there were twenty-six instances of left-handedness, or 27.9 per cent. In the combined groups of epileptic and feeble-minded patients the percentage of left-handedness was 45 as compared with 27.9 per cent. in normal families. In the hope that some light might be thrown on this question by comparisons of the weight of the two hemispheres of the brain, the author studied the brains of 168 cases of epilepsy and feeble-mindedness. Of these, 151 were from those who had been right-handed and seventeen left-handed. In the larger number, the right hemisphere was heavier than the left. There was little difference in this among right-handed and left-handed patients—67.1 and 70.6 per cent.

BARRETT, Ann Arbor, Mich.

EPILEPSIE ET HYSTERIE (EPILEPSY AND HYSTERIA). L. MARCHAND, *Presse méd.* **28**:627 (Sept. 8) 1920.

Numerous and conflicting authorities are reviewed in this discussion of the various relations in which hysteria and epilepsy may conceivably be associated. Tentative conclusions are reached in an endeavor to clarify the prevailing conceptions of the two syndromes. Three questions are answered:

1. In the course of a series of convulsive seizures in a given case, can certain convulsions be distinctly epileptic and certain others distinctly hysterical? Yes; occasionally in traumatic epilepsy, otherwise rarely. Nonconvulsive hysterical manifestations in epileptic patients are less infrequent. However, most patients with so-called postepileptic hysteria would better be classed as pure hysteria.

2. In a series of convulsions, can any seizure be transitional between epilepsy and hysteria, or a combination of the two? No. A so-called combined attack of "hystero-epilepsy" is simply hysteria.

3. Can established hysterical convulsions eventually become transformed into epilepsy? No. Most cases formerly interpreted in this way seem rather to have been epileptic from the beginning, i. e., grand mal had merely been preceded by undiagnosed petit mal. Others belonged in Group 1, suffering from both diseases concomitantly; hysteria disappeared, epilepsy remained. It is also conceivable that epilepsy may have occurred in a person who had already had hysterical convulsions, without there being any demonstrable connection between the two.

HUDDLESON, New York.

A METHOD FOR THE QUANTITATIVE DETERMINATION OF PROTEIN IN CEREBROSPINAL FLUID. W. DENIS and J. B. AYER, Arch. Int. Med. **26**:4 (Oct.) 1920.

The authors have collaborated in a series of tests on spinal fluids with the object in view of ascertaining the quickest and most reliable method of determination of the protein content. Of all methods, they have found that that in which the precipitated protein is compared to a standard of known protein content is best. The precipitating agent used is sulphosalicylic acid, and the standard is prepared by adding to a test tube 3 c.c. of a solution containing 0.3 mg. of protein per cubic centimeter and 3 c.c. of a 5 per cent. sulphosalicylic acid. The protein for the standard is prepared from human blood serum. The unknown fluid is used in a quantity of 0.6 c.c. (except in such fluids as occur in meningitis in which a large protein content is evident—when greater dilutions up to 0.1 c.c. or more are used), adding 0.4 c.c. of distilled water and 1 c.c. of a 5 per cent. sulphosalicylic acid solution. The contents are gently mixed and allowed to stand five minutes. The result is compared by means of a Duboscq colorimeter, with a 30 mm. scale, against the standard solution. Care and a little practice are necessary in the accuracy of the readings. Results in milligrams per 100 c.c. are obtained by multiplying the quotient obtained by dividing the reading of the standard by the reading of the unknown, by 0.3 (when using 0.3 mg. standard), then dividing the product by the amount of fluid taken and multiplying the quotient by 100. Inaccuracies may arise through contamination with blood or micro-organisms, or when the fluids stand a long time uncorked. The method is accurate to within approximately 5 per cent.

PATTEN, Philadelphia.

EXTRACTION D'UNE BALLE SITUEE DANS LE VENTRICULE CEREBRAL LATERAL (EXTRACTION OF A BULLET SITUATED IN THE LATERAL CEREBRAL VENTRICLE). G. L. REGARD, Rev. neurol. **26**:818 (Nov.) 1919.

Regard describes a case in which a bullet was successfully removed by means of a two-stage operation, from the right lateral ventricle, specifically, at the point of division of the body into the posterior and inferior horns. The bullet, migrating from the left hemisphere to this point in the course of three days from the time of injury, had made its entry through the posterior inferior portion of the middle temporal gyrus. The clinical picture was marked by pronounced dimness of vision, myosis, severe headache, feeble tendon reflexes, somnolence and slight elevation of temperature (38.9 C.). The author remarks on the absence of aphasic disturbance, notwithstanding the temporal entry. With the exception of bilateral circular contraction in the visual fields (45-70), and pain on inclining the head forward or continued reading, the patient apparently made a very good recovery, and in seven months was able to resume his former occupation as carpenter.

RAPHAEL, Kalamazoo, Mich.

ACTION OF HORMONES AND ALKALOIDS ON IRIDES OF DOGS AFTER EXTIRPATION OF THE SUPERIOR CERVICAL GANGLION. A. MAZZEI, Arch. di Ottal. **26**:249 (Nov.) 1920.

The author extirpated the superior cervical ganglion on one side in five dogs and tried the effect on various substances on the iris, using the other eye as a control. The substances were administered by the vein, with the

exception of pilocarpin and cocain, which were instilled in the conjunctival sac.

Epinephrin produced immediate mydriases on the operated side and no effect on the iris of the other eye in eight experiments. These results agree with the observations of Meltzer and Auer, and must be due to removal of inhibition which the ganglion exerts on the dilatator irides, allowing the epinephrin to act. Endospermin produced miosis on the operated side and slighter miosis on the other side. Chloral hydrate produced mydriasis in both eyes, more marked on the operated side. Atropin produced mydriasis in both eyes, much later on the operated side. Cocain administered by the vein and by instillation in the sac, produced prompt and complete mydriasis on the unoperated side, while the mydriasis was later and incomplete on the operated side. This effect and that of atropin were due, apparently, to the cutting off of the innervation to the dilator. Pilocarpin instilled in the sac and injected into the anterior chamber, produced miosis on the operated side fifteen to twenty minutes earlier than on the unoperated side. Endovarini, Witte's peptone and sodium nitrite had no effect on either eye.

REESE, Philadelphia.

TROIS CAS D'ATROPHIE MUSCULAIRE CHEZ DES PARALYTIQUES GÉNÉREUX (THREE CASES OF MUSCULAR ATROPHY AND GENERAL PARALYSIS). M. BRIAND and J. ROGUES DE FURSAC, *L'Encephale* **15**:553 (Oct. 10) 1920.

These authors report three instances of muscular atrophy in paretic patients. One was of the Aran-Duchenne type, another of the Charcot-Marie-Tooth type and a third, perhaps neuritis, perhaps an Aran-Duchenne type—the authors are not certain. The first and third patients also had signs of tabes dorsalis. The third patient had a positive Babinski reflex on the left. There are four excellent photographs. The authors discuss the possibilities of diagnosis and conclude that syphilis caused both the mental and muscular signs.

The interesting point is that syphilis may, in the same person, produce disease of the brain (paresis), the posterior roots and columns (tabes dorsalis) and the motor cells and tracts. Cases of tabes with muscular atrophy are not uncommon. The authors show the coincidence of paresis with such atrophies. The coincidence of tabes and paresis is well known. The article serves to emphasize the various combinations occurring in syphilis of the nervous system.

KRAUS, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

*Three Hundred and Eighty-Fourth Regular Meeting,
Scientific Session, Dec. 7, 1920*

WALTER TIMME, M.D., *President, in the Chair*

LETHARGIC ENCEPHALITIS WITH SEVERE RECURRENT NARCOLEPSY. DR. WALTER M. KRAUS.

Dr. Kraus presented a boy who had lethargic or narcoleptic attacks during the day, with insomnia at night. The patient had an influenza-like attack in February, 1920, and after that he was continuously somnolent for six weeks. He could be aroused, and then recognized his family. This somnolent period was followed by inability to sleep at night, and drowsiness during the day. He would fall asleep while standing up. He was admitted to Bellevue Hospital.

Examination revealed an undersized boy of 12 years. He had a peculiar stooping, parkinsonian attitude while standing. When a narcoleptic attack began while the patient was standing up, the head would fall forward, then the torso would flex on the hips and the knees bend, and the patient would fall to the ground if not caught. There was double paresis of the facial nerves of central type, the arms were in the parkinsonian position, the right more so than the left. The fingers were in the pill rolling position. There was no change in sensation. The tongue was thick, the teeth spaced; there was a general cretinoid appearance. Since his visit to the hospital, dribbling at the mouth has been noticed. The rest of the examination was quite negative.

During the presentation the boy began to bend forward, his head drooped on his chest. When Dr. Kraus called attention to his condition he noticed that he was being spoken of, smiled, and straightened up again. His tongue protruded slightly and remained so for some minutes. Photographs taken during the narcoleptic state show him with the body almost doubled.

Combined with day sleeping there is almost total sleeplessness at night. This is the inversion of the sleep mechanism found in many cases of epidemic encephalitis.

The Wassermann reaction was negative of both blood and spinal fluid. The fluid contained 4 cells; the globulin was negative. An estimation of the total sugar in the spinal fluid showed 0.95 per cent. (normal, 0.40 to 0.60 per cent.). The colloidal gold reaction was 0011112110. The urine was normal. Red blood cells numbered 4,900,000; white blood cells 8,800; there was 90 per cent. of hemoglobin and 50 per cent. of polymorphonuclears.

At the time of presentation (at night) Dr. Kraus considered it striking that the attacks were not occurring with the same intensity as during the day. They had been frequent in the ward and had been observed that afternoon.

The case is typical of the group showing involvement of the basal ganglions.

PROGRESSIVE LIPODYSTROPHY. DR. WALTER M. KRAUS.

Dr. Kraus showed a young woman suffering from progressive lipodystrophy. Not more than twenty-five such cases have been reported. At the age of 8 it was noticed that the fat about her face began to disappear and the hips grew heavier. The disease progressed for fourteen years, and when presented there was marked atrophy of subcutaneous fat down as far as the first lumbar segment, with fatty deposits below that point. There was a condition of the legs which appeared to be edema, but the legs did not pit on pressure.

Examination revealed nothing else of consequence. Dr. Kraus suggested that this condition was analogous to the trophedema of Meige. Pathologic examinations in progressive lipodystrophy reveal a subcutaneous accumulation of fat and a proliferation of connective tissue. This is also true of trophedema except that the accumulation may involve both legs only, one leg only or one side of the body. It would appear that in typical progressive lipodystrophy there exists in the lower extremities a condition of trophedema on which is superimposed a further disorder of the subcutaneous tissues manifested as a localized multiple lipomatosis.

In the October, 1919, issue of the *Revue Neurologique*, there is a critical study of progressive lipodystrophy by Boissonas. This author concludes that the cause is not known, but that a spinal cord origin is most probable.

DISCUSSION

DR. WALTER TIMME asked whether any metabolic changes had been noted in the roentgen-ray studies.

DR. KRAUS replied that no such changes had been observed.

AN EXPERIMENTAL STUDY OF THE EFFECTS OF RADIUM EMANATION ON THE BRAINS OF ANIMALS. HALSEY J. BAGG, PH.D.

The work of the Memorial Hospital on radium treatment of tumors of the nerve tissue was reported with lantern slide demonstration by Drs. Bagg, Ewing and Quick. Dr. Bagg said that the primary purpose was determination of a suitable technic for the use of radium emanation in the treatment of brain tumors. The experiments were designed to throw light on three points: first, the nervous tissue reaction of the normal brain after exposure to radium emanation, from a histologic standpoint; second, the question of dosage and safety; third, the most practical method or methods of applying the radiation.

Four methods were used. First, unfiltered radium emanation in minute quantities, 0.2 to 1.5 millicuries each, was permanently inserted beneath the scalp in rats, and directly into the brain tissue in rats, guinea-pigs, rabbits and dogs. Second, unfiltered radium emanation in considerably larger doses, from 63 to 255 millicuries, was inserted directly into the brain substance of rabbits and dogs and left in place for varying intervals of time. Third, a comparatively large amount of radium emanation, filtered by 1 mm. of platinum and in the form of capsule applicator, was inserted into the brain of a dog for thirty-five minutes. Fourth, two large doses of heavily filtered radium emanation, consisting of 4,000 and 9,000 millicurie hours, respectively, were applied externally over the head in dogs, and a still larger dose of 12,030 millicurie hours was applied over the left temporal region of a monkey.

A long, fine, steel trocar was passed through a small hole in the skull and through it a minute glass tube containing the radium emanation was inserted into the brain. The traumatism incident to the insertion of the trocar was slight. A radiograph showed the platinum capsule in place. The capsule was removed by means of an attached fine brass wire. The so-called lead tray that was used in the external applications gave a filtration of 2 mm. of lead, and in addition 0.5 mm. of silver was used. This applicator, in the case of the three treatments that employed heavy external radiation, was held from the scalp at distances of 2, 10 and 50 mm., respectively.

The most interesting results were: First, the characteristic localized radium destruction was the most marked feature of the method wherein small amounts of unfiltered radium emanation were embedded in brain tissues. This effect was accompanied by pronounced polymorphonuclear leukocytic infiltration, which surrounded a completely necrotic area of brain tissue. The amount of destroyed tissue was about 1 c.c. in nearly all cases, generally exactly that amount, seldom less, and never more than 1 or 2 mm. more in diameter.

Second, a considerably greater amount of destruction accompanied larger doses of unfiltered radium emanation, left in the brain for shorter periods of time, although the dose, as judged by the number of millicurie hours, was the same as for the small doses, which acted over a comparatively long period.

Third, comparatively slight, if any, brain changes followed exposure to strong doses of heavily filtered external application of radium emanation, although such doses were considered of sufficient strength materially to affect the cells of a brain tumor.

It is interesting to note that a considerable amount of brain tissue was destroyed by the first method, without the animals showing any discernible neurologic disturbances, even though they were under observation for over six months, but that when the same dose in number of millicurie hours was given by means of a comparatively large amount of radium emanation, acting over a short period of time, the neurologic reactions accompanying this more severe and rapid destruction were pronounced and generally terminated fatally a few days after treatment.

The three large doses of radium emanation, heavily filtered and externally applied, showed that the normal brain, as judged by gross and microscopic examination, as well as the absence of neurologic symptoms, was markedly resistant to exposure to gamma radiation of radium emanation. In the case of the two dogs thus treated, care was taken to protect the scalp, and the animals were apparently well and active at the end of a month. In treating the monkey the radium dose was greatly increased and the applicator was placed near the scalp, thus increasing the intensity of radiation on the skin as well as within the brain. A careful study of the animal's neurologic reaction showed nothing abnormal. The animal had previously been trained, by the behavioristic method, to obtain its food by opening a puzzle-box fastened by three catches, and it is interesting to note that its reactions to the box situation were only slightly different before and after treatment, and the changes that did occur were probably referable to disturbing factors arising from a severe radium burn, which later developed over the side of the head that was exposed to the radiation. The results of the burn culminated after a month's time in the death of the animal, and the study of the brain in this case, although the radiation had been increased to the limit, showed no gross changes, except

a certain amount of anemia of the cortical blood vessels of the brain directly exposed to the radiation; a microscopic examination revealed no definite degenerative changes.

Of the four methods that have been tested, one may consider the surface application of heavily filtered radium emanation as a relatively safe procedure in the treatment of brain tumors. The burying of small doses of unfiltered radium emanation is also suggested as an especially favorable method of treatment. The relatively sudden destruction produced by comparatively large doses of unfiltered radium emanation makes this method a doubtful procedure. While the embedding of filtered radium emanation is still uncertain, it is possible that by using still larger doses than were employed in these experiments, and decreasing the filtration, this method might also be considered applicable.

THE STRUCTURE OF NERVE TISSUE TUMORS WITH REFERENCE TO RADIUM THERAPY. DR. JAMES EWING.

Dr. Ewing said that regardless of its future as a therapeutic agent, radium therapy has demonstrated certain previously unrecognized biologic properties of malignant tumors. Basal cell carcinoma is susceptible to radium, but squamous carcinoma comparatively insusceptible. Lymphosarcoma disappears readily under gamma rays.

The structural characters which determine susceptibility to radiation are of a cellular character, an undifferentiated form of the cells, rapid growth with abundance of mitoses, vascularity, especially when due to abundance of delicate capillaries, and absence of much intercellular substance. When the cells are differentiated and adult in type, when they grow slowly and mitoses are few, when the blood supply is through well formed adult vessels, and when there is much intercellular substance, the tumors are relatively insusceptible.

Neurofibroma or neurosarcoma presents features of the resistant tumor. This is unfortunate since it is so common, and since it is especially prone to recur after excision. More than 100 recurrent cases of this type have been received at the Memorial Hospital during the past two years. Most of these tumors are not recognized and are designated sarcoma. The structure of intertwining fibrils and long spindle cells is quite specific, however. They differ in reaction to radium from the soft and vascular fascial sarcomas. They first occur as apparently innocent movable tumors of the subcutaneous tissue or intermuscular planes. If surgical intervention is not successful, little aid from physical agents may be expected. Neurofibroma of the acoustic nerve has an unfavorable prognosis usually, but there is a myxoglioma of the optic nerve occurring in young subjects, which does not recur after enucleation.

Of the endotheliomas, psammoma, since usually subdural in location, might be affected by radium, especially if the radium is applied directly to the tumor. True angio-endothelioma or peri-epithelioma, composed of large polyhedral or cubical, occasionally flattened cells surrounding blood channels, occurs in the rare diffuse sarcoma of spinal meninges, and should be more susceptible to radium.

Angiosarcoma, one of the few tumors that has been satisfactorily traced to a traumatic origin, should be markedly influenced by radiation, since its nutrition is unstable, but whether slow and safe regression can be accomplished is doubtful, especially with bulky tumors.

Glioma, however, of all tumors of the brain and spinal cord, presents most of the structural features that favor susceptibility to radiation. It is the most frequent brain tumor, and is chiefly cortical. Its comparatively rapid growth, lack of encapsulation and secondary effect on surrounding brain tissue are unfavorable features. Of the three main types, astrocytoma, gliosarcoma, and neuro-epithelioma, only the first contains anything like resistant intercellular material.

Primary carcinoma of the brain assumes an embryonal type of ependymal glioma, or has the adult type of papillary adenocarcinoma. The latter usually have small cells and very delicate mucinous stroma and grow slowly. Their structure indicates a high degree of susceptibility to radium.

The group of hypophysial tumors includes cysts, chronic adenomatoid hyperplasia, cellular adenocarcinoma, glioma and hypophysial duct tumors. There are no data suggesting that radium can affect the accumulation of fluid in the cysts. Chronic hypophysial struma with acromegaly has already been definitely influenced by roentgen rays directed through the temporal regions.

The possibility of applying radium successfully and safely depends on the obstacle presented by the skull and the distance of the tumors from the scalp. These obstacles are met by increased dosage. Effective dosage of radiation can be delivered through the skull to influence the growth of cellular tumors, as has been demonstrated experimentally by the work of Dr. Bagg on dogs and monkeys. In the brains of normal dogs and monkeys a dosage of 2,000 millicuries of emanation filtered through 2 mm. of brass and placed for six hours at a distance of from 6 to 10 cm. from the skin, has not produced any structural changes, although it has produced rapid regression of deep glandular carcinomas, metastases of testicular carcinoma and retroperitoneal lymphosarcoma. A much higher dosage, resulting in caustic necrosis of the scalp, may be tolerated by normal brain tissue. When the tumor can be exposed it becomes accessible to direct application of radium or the insertion of emanation needles. If the latter are to be used, it is important that the tumor tissue should not be disturbed by partial excision.

CLINICAL RESULTS OF TREATMENT OF NERVE TISSUE TUMORS BY RADIUM. DR. DOUGLAS QUICK.

Dr. Quick said that there were few cases that had been studied clinically, and the literature is deficient. Most of the cases to which radium treatment was applied were pituitary tumors. Bieleva, Loeb, Cauvin and Gunsett are the chief exponents abroad. The general opinion is that the pressure symptoms may be relieved to a considerable degree. Pressure symptom cases are extrasellar and therefore usually inoperable. Headache and eye symptoms are relieved rapidly in many cases. When changes are due to cysts, there is less likelihood of favorable results.

There is marked improvement in pressure symptoms with checking of the metabolic and trophic symptoms. The tumor is in a favorable location for treatment. Exposures may be made from the front and sides or from the nasal route. The floor of the sella may be removed giving more direct access.

Of three cases which Dr. Quick has already reported, one patient died. This patient had a pituitary tumor removed some time previously. A second operation had been performed followed by treatment by radium radiation. The patient had profuse coryza at the time of the treatment. Meningeal involvement appeared, and the patient died. The infection had been introduced by the nasal route.

The second case was that of a young woman who had had two operations for adenoma the year before. There were blindness in the right eye, partial blindness in the left eye and projectile vomiting. She was treated externally and by the nasal route. The vomiting and dizziness were relieved. The eye symptoms were stationary. The patient, two and a half years after treatment, is able to get about and is fairly comfortable. Nothing can restore the vision.

The third patient was relieved from severe headache and pressure symptoms. Operative procedure without exposure of the tumor was carried out for exploratory purposes only. The patient returned with severe headache, which was relieved by treatment. Dr. Quick's earlier experience with intracranial tumors was limited and unsatisfactory. One was a cortical tumor in which there had previously been a right temporal decompression. This was radiated with extremely small doses; the treatment was short and the patient died. Another was a tumor in the region of the vermis. Treatment was applied on either side and posteriorly. There was improvement for six weeks, then there were cerebellar ataxia and vomiting; and the patient had to be carried to the office. Six weeks later he could walk. Then the symptoms recurred with increased pressure. A decompression was done, and the child being in poor condition died. The tumor had not been localized. The case of a child of four, who had an orbital tumor, was also cited. The mass in the upper right orbit was removed and recurred. External radiation treatment was applied for five months. The mass could be promptly reduced, but would recur. The eye was then removed, and radiation carried on in the orbital cavity. The tumor was attached to the orbital nerve. The growth was essentially a spindle cell myxosarcoma. It was not entirely removed. Radiation was applied directly to the base. Although only a short time has elapsed since the operation, recurrence has not yet been noted.

In over 100 cases of neurosarcoma at the Memorial Hospital only one was a primary growth, while the recurrences ranged from one to twenty-one operations. This type of tumor is discouraging in general. A given area may be destroyed, and another recurrence may take place somewhere else along the course of the nerve, not necessarily by continuity. It can be destroyed locally if large enough dosage is used.

Dr. Quick said that our knowledge indicates the judicious use of radium and surgery combined. Operation should not be resorted to unless radium is at hand to be applied at the same time if it be found possible to bury it in the tumor.

Two patients were presented at the meeting, the first a boy of 7 who three months before had had frontal headache, projectile vomiting, double vision and ataxia. He had had a convulsion two months before. A decompression was performed, and a neoplasm in the left lateral lobe of the cerebellum and vermis was found. Bilateral decompression was performed which relieved the pressure symptoms and vomiting. He was unable to walk when admitted, was ataxic and fell to the left. There was bilateral and vertical nystagmus. The disk outlines were hazy. Treatment consisted of three applications, of radium, one dose of 8,712 millicurie hours at the left occiput, 8,000 at the right occiput, and 7,000 posteriorly. Ten days after the first dose he was able to sit up and could coordinate better. After the second series of doses his station improved. There is now no evidence of cerebellar disturbance. Epilation of the hair is marked over the radiated area. In all 53,728 millicurie hours of radium were applied, practically all at 6 cm. distance.

The second patient, a young woman of 23, was referred to Mt. Sinai for tenderness in the right lower chest and right scapula. She had been injured six years before. The right leg was stiff and tense and dragging. For three weeks she had complained of girdle-like pains. Roentgen-ray examination showed deviation to the left, and a mass to the right of the fourth dorsal spine. It seemed to be bony and apparently was connected with the vertebral column. A portion of the tumor was removed. The infiltrating base was adherent. Eleven days after operation the patient was sent to the Memorial Hospital. The tumor tissue was found to be composed of polyhedral and spindle cells, chiefly nuclei. The tumor was classified as an anaplastic glioma. Its origin from ependymal cells was considered possible.

A large dose of radium was applied to the site of the tumor. Packs at a distance of 10 cm. with a dosage of 18,000 millicurie hours were first used; twelve days later, 11,000 at 6 cm. She was then able to move her legs more freely. Four weeks later, three doses of 6,000, 7,000 and 12,000 millicurie hours were given. In all 60,378 millicurie hours were applied, all except the first dose at a distance of 6 cm. She now has control of her extremities to a large extent. The improvement has been gradual but is pronounced.

The amount of radium found necessary for use externally was 50,000 or 60,000 if filtered. It was found relatively safe to use radium directly in the substance of the brain. Therefore exploratory operation and radium therapy should be carried out at the same time, and should be provided for in every case. In the boy's case much less radiation would have been sufficient, and the result would probably be more permanent.

DISCUSSION

DR. CHARLES H. FRAZIER of Philadelphia (by invitation) said that his interest in the effect of radium on tumors of the central nervous system was first aroused in 1914. He called attention to a number of points in which malignant tumors of the brain differed strikingly from malignant tumors in other parts of the body and why there was a larger field for radium in the brain than in other organs or structures. Much information in matters of technic must be derived from the experimental work. Dr. Frazier's experience included twenty-five cases, of which number six were gliomas, six endotheliomas or sarcomas, one teratoma and twelve undetermined. He said that pituitary lesions lent themselves to radium therapy because of the opportunities, after a sellar decompression, to place the radium tube in close proximity to the lesion. He believed the first step in the surgery of pituitary lesions was a simple subsellar decompression without removal of tissue, except perhaps for diagnosis, this operation to be followed by roentgen-ray treatment to be repeated at such intervals as seemed desirable.

As to the results obtained, in the total series, eleven of the twenty-five patients have died since the treatment began, and there are fourteen still living. Of the survivors, three are alive six years after treatment; one is alive five years after treatment; four are alive three years after treatment; two are alive two years after treatment, and four are alive one year after treatment.

Of this number, there were four in whom the tumor was removed and two of these are known to have recurred. Of the latter, one is a sarcoma of the right cerebellar hemisphere which is being treated by direct implantation of radium needles. After the last treatment there was, within two weeks, a substantial improvement.

In analyzing the results he found that the best work had been obtained in pituitary lesions and in endotheliomas. He has no evidence that radium has any influence on gliomas. The series which he has reported should not be regarded as a criterion because it has been impossible always to apply radium at such intervals as seemed desirable. Patients living at a distance from the hospital often could not be persuaded to return when requested to report. As to the conclusions, the evidence at hand justifies the following statements: 1. Radium will retard the growth of endotheliomas; two patients still under observation after six years. 2. There is no evidence warranting the assumption that radium influences the course of the gliomas. 3. Radium has proved effective in lesions of the pituitary body. Radium is now employed: (1) as a prophylactic against recurrence always after the removal of the growth; (2) as a prophylactic against recurring visual disturbances after sella decompression; (3) as an active agent by direct implantation in all inoperable growths exposed on the operating table; (4) as an inactive agent by indirect application in all inoperable growths.

DR. H. K. PANCOAST of Philadelphia (by invitation) said that there are many reasons for regarding tumors of the brain as an admirable field for treatment by radiation. In order to determine the effects on normal brain tissue it was necessary to carry out preliminary experiments on lower animals, and dogs were selected for the purpose. Over a year ago, under the direction of Dr. Frazier and himself, some third and fourth year medical students took up the work of implanting radium tubes in dog's brains and studying the effects after a period of time which should have been sufficient to show any changes. After from four to five weeks following the implantation the dogs were killed and the brains removed and sections made of the areas treated. The exposures were made over the motor area in order that any resulting motor disturbances could be observed in addition to any visible effect on the brain tissue. They started with doses of 300 mg. hours, and the doses were increased up to 900 mg. hours. One tube of 50 mg. of radium, well filtered in order to remove all beta rays, was employed in each instance. The tube was placed directly on the motor cortex.

The results of these experiments have already been published, but brief mention will be made of the results in order to compare them with the results of other experiments by Drs. Pancoast and Frazier and others.

Microscopic sections of the brains of the dogs exposed to 900 mg. hours showed that for a distance of 2 mm. approximating the tube there was necrosis of brain tissue. Outside of this there was an area 2 mm. wide in which the blood vessels showed endarteritis, and for a distance of another millimeter there was a zone of hyperemia.

These experiments assured them that comparatively large doses of radium could be employed in the treatment of brain tumors without danger of serious destruction of brain tissue surrounding the tumor; in other words, in using a reasonable dosage of radium the effects of the radiation on the surrounding normal brain tissue can be disregarded. Other experiments were recently carried out by Dr. Pendergrass, assisted by some fourth year medical students, in order to determine the effect of larger doses of radium on the motor cortex and of implantation of radium into normal brain cortex. Two dogs were used. In one a dose of 2,100 mg. hours and in the other a dose of 2,500 mg. hours was applied over the motor cortex directly on the surface. Both dogs died in about one month. There was no paralysis and no necrosis of the flaps. The adjacent brain area was found to be congested and indurated.

Death probably resulted from the effect on the brain, but sections have not yet been made. These experiments show that there is a limit to the direct exposure which can be applied to the brain of an animal the size of a dog.

Further experiments were carried out by implantation of two needles containing 25 mg. implanted in the motor cortex of two dogs for eighteen and twenty-four hours, respectively, giving a dose of 450 and 600 mg. hours. Paralysis did not result. Both dogs died suddenly within a week, but were well two days before death. Necropsy revealed no infection of the flap or the brain. There was a general serositis, including the peritoneum, pleura and joints. The peritonitis was of a virulent form. Unfortunately, cultures were not made. The needles were carefully sterilized in alcohol for one hour before implantation, which would seem sufficient to bring about sterilization. These experiments are not conclusive.

In one experiment carried out by Dr. Bagg, in which a large dose of 4,000 mg. hours was applied outside of the skull and the brain was exposed by cross-firing to that large dosage, no subsequent microscopic changes in the brain tissue were found. Much valuable information can be gained from these experiments, and it is quite possible to base a fairly safe technic on them.

Treatment of tumors of the brain by radiation requires careful observation of these points: (1) knowledge of the type of growth to be treated; (2) knowledge of the relative amount of radiation required in the treatment of such growth; (3) size of the growth from the center to the periphery where the tumor is actively proliferating if central implantation is practiced; (4) if the growth is irregular and not truly spherical, there is still greater reason for carefully guarding against undue effects on normal brain tissue; (5) it is best to give an underdose by implantation and to supplement this by cross-firing from the outside of the head.

Drs. Frazier and Pancoast are as yet unwilling to advocate implantation of needles at the actively growing periphery of tumors until the action of radiation on brain tissue is better understood, and the margin of safety has been better demonstrated.

Tumors may be grouped in the following manner for treatment:

1. Tumors found at operation and which cannot be removed. A moderate dose of radium may be applied by implantation of the tube in the center of the growth, and this supplemented by cross-fire radiation from the outside of the head.

2. Tumors found at operation and partly removed. These may be treated in the same manner.

3. Tumors not localized and not found at operation or inaccessible at operation. Such tumors may be treated by cross-fire radiation from the outside of the head in all possible directions. This may be done apparently without any danger to normal brain tissue.

4. Pituitary growths may be treated after decompression, after partial removal or at a time subsequent to operation when there is a recurrence of symptoms. The technic employed by Drs. Frazier and Pancoast has been the application of radium below the sphenoid sinuses for a period not sufficient to cause serious damage to the structures in this neighborhood. This is supplemented by cross-fire radiation with roentgen rays through each temporal region toward the hypophysis.

DR. ROBERT ABBE (by invitation) said that during eighteen years of earnest study and use of radium in every surgical condition, he had had only three

cases of cerebral or spinal tumor referred to him for treatment, and these were postoperative, in which the gliosarcoma had been impossible to remove and the wound was closed over them. The bone, however, had been removed by trephine and rongeur so that radiumization could be given (through an inch thickness of fleshy tissue). No benefit had followed that he could see.

Dr. Abbe believed that the better technic would be to apply radium (25 or 50 mg.) in a small sterile tube without filtration of metal, directly to the tumor in the open wound under anesthesia for thirty minutes; then close the wound completely. His experience with this method in true cancer in other parts of the body, as suggested by Dr. Wickham of Paris in the early days of radium work, had given him more wonderful results than by any other method.

To some degree the skin seems to act as an insulator against penetration of the best soft beta rays which are the most fruitful of results. In fibrosarcomas of the dura he had found no benefit from radium. He had never seen cerebral disturbance from radium applications externally in human subjects.

Dr. Abbe had treated four cases of sclerosis of the cord (syringomyelia) referred to him by neurologists. In three cases no benefit followed. One case showed extraordinary beneficial results. The patient had led an active life until eighteen months before Dr. Abbe saw him. Then an ascending hemiplegia began in the leg and arm, with other characteristic symptoms, eliminating a cerebral lesion. Exhaustive study by the eminent men of the Neurological Institute gave no hope. When seen by Dr. Abbe the patient could walk two blocks with unsteady gait by aid of a cane. The arm as well as the foot had spastic paralysis, with cramps.

Radium, 250 mg., shielded by 0.1 mm. of lead, and 2 inches of gauze, was applied for from two to four hours at a time at suitable places on the spine, from the occiput to the midlumbar region, once or twice a month. In six weeks he showed improvement, and in three months he walked 4 miles daily. Later he gave up his cane and walked 10 miles daily in his business. His spasms and paralysis improved markedly, and he has had no relapse up to date, four years after treatment. He uses his paralyzed hand to cut his food and turn the key in the door. No other medication or treatment was given. The Wassermann reaction was negative.

DR. ISRAEL STRAUSS said that direct radiation of tumors at the surface and in parts of the brain where removal would incapacitate the patient, would be advantageous. In such cases surgeons should *not* remove the tumor and then apply radium. In the boy shown by Dr. Quick, the tumor was in the left lateral lobe of the cerebellum and vermis. At operation the vermis was enlarged, gelatinous in appearance and displaced to the right. It would have been risky to insert radium. It was considered far better to carry out the decompression and then to turn the patient over to the radiologists. Not a symptom of tumor is now present, but it is too early to state whether the results were attained by the radium or by the decompression.

In a case of pituitary disturbance observed some four years ago, applications were started, after sellar decompression, in the nasopharynx. The radium was put into the tumor. This was a mistake, for meningitis resulted in forty-eight hours. The procedure should have been sellar decompression to save the eyesight, then radium applied in the nasopharynx. Enough radium should be used to go through the sphenoid. The removal of the floor of the sella turcica offers an opportunity for herniation. One patient in whom there had been great improvement for a time, showed return of symptoms; a mass was

found in the nose which was subjected to manipulation with resultant streptococcus infection and death. Dr. Ewing thinks radium, if used early enough, is sufficiently penetrating to use without sellar decompression.

Glioma ought to be susceptible to radium. Surgery does not cure glioma of the brain in most instances, since it is so difficult to get at, so difficult to remove, and since it usually recurs. Radiotherapy, therefore, ought to be tried and may, when we know more about it, furnish a means of treating these growths successfully.

DR. I. ABRAHAMSON said that it was important to ascertain the effect of radium on the choroid plexus and cerebrospinal fluid.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Dec. 16, 1920

JOHN J. THOMAS, M.D., *in the Chair*

The program was presented by members of the staff of the McLean Hospital.

APPLICATION OF THE RATING SYSTEM TO NURSES' NOTES. DR. JAMES S. PLANT.

Dr. Plant considered that nurses' notes at present are not objective enough; they cover the same field as the medical notes and have practically no final determinative value. They should be entirely conduct notes. He described the application of a rating system to nurses' notes as follows: By dividing the entire conduct field into some eighteen categories and giving in each category five ratings, a set of ninety fairly simple, definite, objective conduct entities are given the nurse, who has only, in each category, to give the approximate rating. Number 5 in each category fits the isolated ego—4, 3, 2 and 1 representing increasing adaptation. The resulting average represents a "coefficient of sociability." If for recovering and clear cases there is a high coefficient of correlation between the various ratings, the standard deviation may give us insight into the amount of confusion present. The difficulties seem large and are dependent for their solution entirely on experience. The mathematical implications are far-reaching and interesting. The chief danger lies in becoming too mathematical—in arriving at figures and curves so sophisticated as to fall of their own weight.

DISCUSSION

DR. DONALD GREGG stated that in private hospital work he considered it important to have full nurses' reports, and that he had worked out a chart which gave opportunity to record not only the conduct morning, afternoon and evening, but the occupation of the patient as well. In this way the nurse is given a chance to express how she is helping the patient, and it is more interesting for her. This record of occupation day by day gives a good idea of the progress of the patient.

DR. H. I. GOSLINE said that a study of a somewhat similar nature was made at Danvers in 1914, except that the nurses' notes were not controlled by categories. It was found that the terms the nurses used were often much

more objective than those of the physicians. The latter were apt to use terms allowing several interpretations and which expressed not what they saw but what they thought they saw. It was found that symptoms classed as hyperkinetic over a period of years often became hypokinetic in the same patient. This was expressed mathematically in the study referred to.

DIAGNOSTIC DIFFICULTIES IN THE EARLY STAGES OF MENTAL DISORDERS. DR. THEODORE A. HOCH.

DR. HOCH stated that psychoses in their early stages are subject to marked variations in symptomatology, depending partly on the personality, the causative agents and on the environmental factors. The psychotic picture may also be given a certain twist by accidental happenings of an emotional character. Thus an acute psychosis may be colored by dementia praecox-like reactions, or atypical cases of dementia praecox may have a predominating tone of depression. The presence or absence of hallucinations or delusions is of less significance than the mechanism back of them. Diagnoses cannot be made on the presence of isolated symptoms but every obtainable clue, past and present, relating to the patient and the psychosis must be carefully studied. Even then the difficulty at times is cleared up only after months of observation or after recovery. Peculiarities in conduct, eccentricities, suspiciousness, difficulties in making adjustments and other oddities in the make-up of the person are carried into the psychosis when it appears and must be given their proper place in considering the diagnosis. Depressed emotional states, delusions, hallucinations in any field, silly affect or periods of exhilaration may be found in bewildering array and surprising combinations in the formative stages of mental disorders. The greatest difficulty naturally lies in differentiating dementia praecox from manic-depressive insanity, and at times even careful analysis of every factor may leave the diagnosis in doubt.

DISCUSSION

DR. ARTHUR H. RUGGLES considered Dr. Hoch's paper valuable especially in that it indicated the difficulty in differentiating the manic-depressive and dementia praecox cases. He emphasized that, while the study of the personality and the grouping of the person according to his personality and environment is of the greatest value in trying to determine the type of psychosis, too much confidence cannot be put into making the diagnosis purely from the personality. The type of personality is almost always bound to appear in the psychosis but does not always determine the psychosis. Dr. Hoch's paper tends toward making the psychiatrist put emphasis on the combination of personality, plus environment, plus the descriptive study of the condition that is under treatment. It will reinforce the conclusion that there are certain cases that must be held without diagnosis, for often, as Dr. Hoch has said, only time and the results of the case will give the correct diagnosis.

THE "CLINICAL" PSYCHOLOGIST. DR. F. L. WELLS.

Dr. Wells stated that there is a type of psychologic work which is appropriate for the management of certain clinical and social groups. These groups include some medical and some nonmedical cases. This field of work is not now covered by ordinary medical training, nor, save exceptionally, by medical men. It is being covered more and less well by persons more or less competent in

psychology. It is the desire of responsible psychologists to organize the training in these lines of work. That they are not parallel to medicine is perhaps evidenced by their occasional collisions therewith. This at least rationalizes an effort with the conscious purpose of bringing these related spheres into better definition and harmony. Is there any part in the diagnosis or management of a person's adjustment problem which, being particularly contributed by psychology, can to advantage be handled by the psychologist? If not, no professional standing belongs to clinical psychology. If there is, it will be well to accord such standing as will keep its activities in proper limits toward society and the medical profession.

The question is raised whether intelligence examinations should be functions of the medical man or rather associated with a type of training that is non-medical. The latter situation is one that seems to be working itself out in practice, both within and outside of medical direction. Facility with these methods is gained only with much practice and quickly lost if not continually exercised. The time required is no little tax on the busy practitioner. The required portion of the medical curriculum is not exactly inviting additions to itself. The standard examinations can be made with considerably less training than is required of the physician, so that it seems doubtful economy for him to undertake them on the considerable scale which alone gives facility. These considerations weigh heavily toward the conclusion that "intelligence" measurement, like the Wassermann test, is a task for delegation to technologists specializing in that class of work.

In contact with the fields of law and medicine, but little assimilated into either, is a province dealing with behavior adjustments of the personality to its surroundings. With such problems, for example, the Judge Baker Foundation deals, and its director can assure you that the medical problem—that is, the problem with which medical education specially fits one to deal, is in most cases absent from these. It means something additional to what medical training has hitherto envisaged, the analysis of environmental and conduct history, personal capacities and tendencies. To meet it, the physician must super-add psychologic experience to his medical education, or work under conditions in which the judgment of the psychologist is available. Such problems do not come to the primary attention of those practicing or attempting to practice psychology as a profession. No competent consulting psychologist will neglect to provide for the medical factor by proper medical means. But where the controlling problem is psychologic, persons of the stamp of Thorndike, Scott or the Hollingworths seem not incompetent to have charge. Such persons may be expected to know and abide by the limits within which their competence adjoins the proper responsibilities of the physician. The value of a profession for individual practice depends essentially on what it can do to get somebody out of trouble. Clinical psychology has developed along this line; touching medicine at feeble-mindedness, the law at delinquency, though in these cases the person is brought to the psychologist rather than going to him. If psychology is to develop further as an individual practice, it must show itself able to take care of a certain class of human troubles better than they can now be met by the aid of the lawyer or physician.

DISCUSSION

DR. C. MACFIE CAMPBELL spoke of the tendency in the psychiatric service to look on the clinical psychologist with a great deal of skepticism. It may be feared that the motive is not altogether generous. It is quite true that clinical

psychologists make mistakes, but they are perhaps not more numerous than those made by the medical profession. The clinical psychologist may be a useful collaborator in many tasks which medical training does not especially prepare for and which the demands of specialized medical work do not allow time for, so that it is rather a dog-in-the-manger attitude to be too suspicious of the clinical psychologist and to overemphasize the fact that there are some who are untrained and incompetent.

It is fortunate that the American Psychological Association has itself taken up the question of standards for the clinical psychologist, so that the medical profession may be able to have some sort of guide to the personnel and will know that, if collaboration is wanted along certain lines, a group of men can be found whose training is more or less guaranteed.

DR. F. L. WELLS, in concluding the discussion, read the proposed law defining consulting psychologists.

"Any person shall be held as a consulting psychologist who shall make a practice of diagnosis or evaluation of mentality of special traits or abilities of supposedly normal human individuals, and who shall analyze and describe mentality with a view to determining mental status; or who shall make diagnosis of degrees of mental defects of individual human beings and make classifications upon such diagnosis; and who shall profess under that title to give expert advice regarding the educational and vocational treatment of both normal and abnormal individuals, or who shall publicly profess to be a consulting psychologist."

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 17, 1920.

SAMUEL LEOPOLD, M.D., *President pro tem.*

A CASE OF SCAPULOHUMERAL MYOPATHY. Presented by DR. ALFRED GORDON.

A middle-aged woman, about five years ago began to have difficulty with her right shoulder. There was no pain. She was able to work with her right arm, and she could elevate and rotate the arm, but there was awkwardness in performing these acts. Gradually weakness of the muscles surrounding the shoulder joint developed, and the patient found it difficult to raise the arm and shrug the shoulder. There was no objective or subjective disturbance in the parts affected. The condition has grown progressively worse. Dr. Gordon said that at present the atrophy was pronounced at the shoulder girdle, the pectoralis major, supraclavicular muscles, supraspinalis and infraspinalis, the rhomboid and the serratus showed considerable wasting. The scapula was displaced, its lower angle being elevated considerably. The muscles of the arm were fairly well preserved but those of the forearm and of the hand were somewhat atrophied. All the deep reflexes of the upper extremity were abolished. Moreover the supraclavicular musculature on the left side had commenced to be involved, and the calf muscles on the right were flabbier than those on the left. Close inspection of the face showed a slightly greater fulness on the left side than on the right. Fibrillary contractions, increased mechanical irritability and reactions of degeneration were all absent in the atrophied muscles. The

knee jerks were normal and equal on both sides. Sensation was normal. Roentgen-ray examinations of the various articulations of the upper extremities have been negative.

The case seems to be one of myopathy of the scapulohumeral variety with the possibility of it being of the Déjerine-Landouzy type.

MYOTONIA ACQUISITA. Presented by DR. N. W. WINKELMAN.

Two cases were shown. The first patient, an Italian boy, aged 12, complained of stiffness of the extremities especially marked in the morning and becoming less as the day progressed. The stiffness was noticed in the beginning of muscular movements, especially in ascending stairs. He was unable to walk or run at first, due to stiffness of the legs but the stiffness gradually wore off until he was able to move about in a normal manner. His mother was insane and died some years ago. He was a seven month's baby, instrumental delivery. At 2 years of age he had convulsions which lasted for a few months. The boy said he was called "The Little Giant" by his comrades.

On examination the boy appeared to be well developed, the muscles stood out prominently, much greater than was usual for a boy of his age. All muscular movements were done stiffly at first but this wore off as was especially evident when the patient walked upstairs. At first he walked like a patient with paraplegia, but after climbing about forty steps he walked in a normal manner. The genitals were small; the hair and the skin were normal. There were no feminine characteristics. The neurologic and general examinations were negative. The reflexes were decreased at first, but as the tendons were repeatedly tapped, they became normal. The electric reactions were of the typical myotatic type. All metabolic and laboratory tests were negative except for albumin in the urine.

The second patient, a boy of 16, was well until 1918, when he had influenza. Since that time he has had difficulty in performing muscular movements because of intense stiffness, which gradually wore off as the muscular movements were continued. The stiffness never entirely disappeared. He noted an increase in the size of his muscles recently without any cause. He had noticed that when he took hold of an object he had difficulty relaxing his grip.

At times there develops a stiffness of the tongue when talking fast and occasionally a stiffness of the muscles of the mouth when eating.

Examination.—The patient was tall, well developed, with muscles above the average size. Even the muscles of mastication were very prominent. All muscular movements were at first practically impossible because of intense stiffness, which gradually relaxed, and as the movements were repeated the stiffness lessened. He showed marked perseveration in the hands and at times in other muscles, notably the eyelids. There were fibrillary twitchings all over the body. The deep reflexes were impossible to elicit because of the spasticity. They were no sensory disturbances and no increase of myotatic irritability on tapping the muscles. The electric reactions were not typical of myotonia.

The classification of these cases under the title of "myotonia acquisita" accords with the ideas of Jacoby of New York and does not agree with the idea of Oppenheim that all these cases are congenital, and that it takes some exciting cause to bring them to the surface.

DISCUSSION

DR. CHARLES K. MILLS said that he had reported one or two remarkable cases of myotonia in the International Clinics many years ago. The muscles of one patient would lock while he was walking down the ward and on several occasions he had fallen backward injuring his head. Dr. Mills thought that observations made by Kinnier Wilson and himself indicated that in most of these cases a lesion of the cerebral or cerebrospinal tonectic apparatus was present, rather than that they were affections, either myopathic or, as had been indicated by some, myelopathic. Undoubtedly the muscles may become affected secondarily. Dr. Mills had reported another case some years ago in a paper on "Clinical Problems of Cerebral Tone." This patient had been in the marine service and had a remarkable condition of perseveration which was shown just as had been illustrated here by the difficulty in unclenching his fist. The man's skull was trephined on the side opposite to the perseveration, and an area of softening was revealed in the midfrontal region, the region assigned by Kinnier Wilson as the seat of lesion in some cases of perseveration.

REPORT OF TEN CASES OF HEMIANOPSIA DUE TO OCCIPITAL INJURIES. Presented by DRs. S. D. INGHAM and H. W. SCARLETT.

This paper will appear in full in the *Archives*.

Ten case histories of hemianopsia due to occipital injuries, together with the perimetric charts of the visual fields, and charts extending ten degrees from the fixation point, were presented.

The literature of the work done on this subject during the war by Holmes, Lister, Riddock, Morax, Moreau and others, was reviewed and compared.

The salient points were: 1. The cortical representation of the macula is located in the posterior part of the visual area at the tips of the occipital lobes. 2. The periphery is represented in the anterior part of the visual area, while concentric zones from the macula to the periphery are represented in that order from behind forward. 3. Areas of the retina along the horizontal axis are projected into the calcarine fissures, while areas along the vertical axis are located on the mesal surfaces of the occipital lobe. 4. Fixation with normal vision may be retained with complete loss of one occipital lobe. 5. The fovea must be considered as an area rather than as a point, with slight overlapping of nerve fibers, thus permitting the retention of fixation and normal vision in cases of complete loss of one occipital lobe.

A case of paracentral scotoma, and one of panoramic loss of vision with retention of only fixation, were presented. The latter had 20/40 central vision in each eye, but no peripheral vision.

DISCUSSION

DR. CHARLES K. MILLS said that years ago he described a case in his book, in which there was what appeared to be a macular hemianopsia. He believed it was the first time that macular hemianopsia had ever been recorded, although Wilbrand perhaps referred to the same thing a year or two later.

HEMIPLEGIA FOLLOWING CHILDBIRTH AND PELVIC OPERATIONS. Presented by DR. A. M. ORNSTEEN.

Four cases were reported. In the first eclampsia suddenly developed in the eighth month of pregnancy, followed in about eighteen hours by a right-sided hemiplegia with aphasia. In the second a right-sided hemiplegia with aphasia and right lateral homonymous hemianopsia occurred two weeks after a still-birth. The blood Wassermann reaction was positive. In the third a left hemiplegia occurred in a syphilitic woman, without loss of consciousness, three weeks after a complete hysterectomy was performed. These three cases were nonfatal. The fourth woman was found roaming aimlessly about the streets two months after childbirth, a victim of puerperal insanity. A week after admission to the hospital she became unconscious and developed a right-sided hemiplegia with aphasia and right lateral homonymous hemianopsia, followed in several days by death. Necropsy revealed a thrombosis of the left anterior and left middle cerebral arteries extending down into the internal carotid artery; softening and degeneration of the entire left hemisphere with the exception of a portion of the posterior part; multiple miliary abscesses throughout the cortex; patulous foramen ovale, cardiac leaflets free of vegetations.

A cerebral hemorrhage, so often seen in fatal cases of eclampsia, was probably the cause of the hemiplegia in the first patient. Whether the lesion in the left middle cerebral artery in the second patient was caused by a syphilitic thrombosis or by an embolus arising from a pelvic thrombophlebitis and passing through the pulmonary capillary circulation, is a diagnostic question difficult to decide. Again, in the third patient, was it syphilitic thrombosis or a postoperative embolus passing through the capillary plexus of the lung in order to reach the left side of the heart and to go thence to the brain? The presence of miliary abscesses in the brain of the fourth patient makes it most probable that a pelvic infection existed, and emboli arising from this region passed through the lung to the brain, where they acted as foci for secondary thrombosis. The absence of cardiac vegetations further strengthens this view. Did they pass through the lung capillaries or through the patulous foramen ovale?

The possibility of an embolus passing through the capillary plexus of the lung, and the uncertainty in pathologic diagnosis in this type of case caused the writer to review the literature for similar case reports and opinions of the respective authors.

Sir James Simpson, in 1847, suggested that all puerperal cerebral emboli arose from cardiac valve vegetations. Seven years later he added five other etiologic views: (a) escape of recently formed unorganized masses of coagulated blood from the heart, (b) a true arteritis, (c) certain diseased conditions of the blood, or certain morbid matters carried by the blood current, (d) laceration of inner coat of vessel, (e) pus or fibrin arising from puerperal phlebitis acting as a nucleus for coagulation. (This latter view must carry with it the belief that the pus or fibrin passes through the lung in order to reach the brain.)

In 1873, Thomas' theories were: (a) spontaneous coagulation in the left heart due to the hyperinotic condition of the blood in pregnancy and puerperium, (b) embolus from a uterine sinus passing through the lung and growing larger by accretion. Rindfleisch and other German pathologists admitted this possibility. Lush refused to accept the theory, and placed the blame on spontaneous left heart clots. Rusch, in 1877, believed that tiny particles from an early mural thrombus passed through the lung and lodged in the brain and by secondary thrombosis caused occlusion of the vessel. Spontaneous thrombosis of the

cerebral vessels was thought to be the cause by Scougal in 1877. Only one other case of extension of the thrombus into the internal carotid could be found in the literature. Angus McLean, in 1916, stated emphatically that an embolus could not pass through the lung unless there was a communication between some of the branches of the pulmonary veins and arteries other than by means of the capillary plexus or through a persistent ductus arteriosus. According to DaCosta, pulmonary emboli may disintegrate and smaller emboli pass to the left side of the heart. Osler states that puerperal cerebral embolism is caused by cardiac vegetations or to formation of clots in the heart because of the increased coagulability of the blood, or from emboli arising from the region of the pulmonary veins.

About thirty-five cases of puerperal cerebral embolism appear in the literature, but most of the reports are unaccompanied by opinions as to the mechanism of their production. The camp today is still divided on this subject of emboli passing through the lung capillary plexus, as it was in the past. In all probability, most of the etiologic factors expounded in the foregoing are operative in certain cases. From results of experiments made by competent workers, it seems that emboli which have lodged in the brain can arise from the venous side of the circulation and pass through the lung.

DISCUSSION

DR. N. W. WINKELMAN said that in the case at Blockley there was a massive clot in the internal carotid extending into the anterior and middle cerebrals and a patent foramen was found. Dr. Winkelman considered this important; he thought quite a clot could go through the foramen ovale because an ordinary probe was admitted with ease.

DR. ORNSTEEN said that Landis, in Norris and Landis' "Diseases of the Chest," states that in a majority of cases a small probe can be passed through the foramen ovale, as in the case reported, and that such a finding need not be looked on as abnormal. The fact that the channel runs obliquely through the auricular wall or septum and that therefore the openings of the two sides are not directly opposite to each other favors competency. These facts, Dr. Ornsteen believed, belittled the probability of an embolus passing through the supposedly patulous foramen ovale.

VARICOSITIES OF THE SPINAL CANAL. Presented by DR. THOMAS E. SHEA.

The specimen presented by Dr. Shea was removed from a body which had been sent to the Daniel Baugh Institute of Anatomy of the Jefferson Medical College for dissection; the diagnosis was cerebrospinal syphilis.

The examination of the spinal canal revealed a varicosity of the internal vertebral plexus of veins which extended from the last thoracic to the sacral vertebra. This, on examination microscopically by Dr. Crawford of the Jefferson Hospital laboratories, revealed a thrombosis of the veins which contained sarcomatous cells and were part of a sarcomatous infiltration that extended from the right testicle to the right suprarenal glands, both of which were sarcomatous. According to the history, the symptoms were mainly of a sensory character and no doubt due to pressure of the spinal nerve roots by congested intervertebral veins. The history also showed that unusual cerebrospinal pressure was present, which accounted for the cranial nerve involvement which the author claimed was due to the pressure of the cranial nerve roots between the base of the brain and its bony bed.

Book Reviews

THE OXFORD MEDICINE. By VARIOUS AUTHORS. Edited by HENRY A. CHRISTIAN, A.M., M.D., Hersey Professor of the Theory and Practice of Physic, Harvard University; Physician-in-Chief to the Peter Bent Brigham Hospital, Boston; and SIR JAMES MACKENZIE, M.D., F.R.C.P., LL.D., F.R.I., Consulting Physician to the London Hospital, and Director of the Clinical Institute, St. Andrews, Scotland. In Five Volumes. Volume 1: The Fundamental Sciences and General Topics. Volume 2: Diseases of Bronchi, Lungs, Mediastinum, Heart, Arteries, and Blood. Cloth. Price. \$62.50 for set. New York: Oxford University Press, 1920.

Under such editorship and with such a list of contributors as is presented, one is justified in expecting a work that differs from the conventional type. Ordinarily works of this type occupy a mid-position between a textbook and such larger works as Allbutt's or Osler's System. They follow conventional lines, try to avoid both extremes of too much and too little detail, and generally are nothing more than this. They rarely stimulate thought. In all of them certain articles are good and others are poor.

In reviewing this first volume one may ask why certain articles are included; but they are without exception interesting and stimulating. For example, one may ask why seventy-five pages on "Aviation Medicine" are included in a work on general medicine. The article covers the qualifications for air service, methods of examination, accidents, experimental work on aviators and the like; all interesting, but why give up space to this subject in a five-volume work on medicine?

Barker has a long article on the "Rationale of Clinical Diagnoses," readable as all of Barker's writings are and far more justifiable than the article on aviation, but one may be forgiven for contrasting it with Trousseau's lecture on "What Is Clinical Medicine," published sixty years ago.

Christian, in the introductory article on "Present-Day Medicine," speaks of the increasing appreciation of the importance of determining the functional capacity of an organ, yet his article on "Tests of Function" is so sketchy as to cover less than ten pages.

The articles so far mentioned illustrate one of the great difficulties in the preparation of a work of this sort; i. e., the distribution of space. One admires the courage of the editors who undertake a job which they know will satisfy no one, not even themselves. But why are ten pages given on what is admittedly the most important present-day task of medicine, and long articles on aviation, eugenics and hydrotherapy, which have no, or scant, place in a five-volume work?

The first volume is announced as being devoted to fundamental sciences and general topics, and it may be said that none of the article fails to come under this classification, but it is probable that about 10 per cent. of the subscribers will be most enthusiastic because of their interest in the wider aspects of medicine, while the other 90 per cent. will be dissatisfied because of the want of specific, simplified statements.

It is noted that while in volume 1 this system is said to be composed of five volumes, in volume 2 it is said to consist of six. This is an important

notice, for if correct, it justifies the first volume, in which far too much space was given to general topics, which, however important and however well handled, limited too much the room in a five-volume system for the proper subject matter of a work on medicine. In five additional volumes this can be adequately handled.

This volume covers the bronchi, lungs, mediastinum, circulation and blood. Without exception the articles are thoroughly good and such of them as have broken away from the conventional method of treating these subjects are notably good. No doubt some will criticize the sections on treatment, but to the reviewer they appear exceptionally good because they are confined to well established rational methods, omitting the useless list of therapeutic agents that have no claim on our attention other than that of old and not very respectable acquaintances.

Hoover's chapter on diseases of the bronchi is handled in just the unconventional way in which one might expect, and in reading it one can almost hear him talking in his calm, well-balanced, hard-headed way. Considerable space is given to a necessary consideration of the physiology of the bronchi and respiratory movements. One wonders why so high a percentage of his space is given to certain subjects, for example, that given to congenital malformations of the bronchi; but this is a matter of judgment on which there is room for legitimate difference of opinion.

A brief discussion is given on the effects of excoriating gases on the respiratory tree, sufficient and well put, but one may ask whether the late effects of gas poisoning are to be considered elsewhere, for most practitioners are now interested in these late effects as all are occasionally seeing ex-soldiers who are, or who claim to be, suffering from the effects of gases inhaled two or three years ago. The reviewer would be glad to know what Hoover thinks of these cases, as he saw many of them during the acute period.

It will be a comfort to many to know that Hoover has never been able to make a diagnosis of bronchiectasis except on inferential grounds, as it will be a shock to some who make this diagnosis readily and fail to realize that the diagnosis is inferential and not on evidence. Every one should read the sections on the movements of the costal margin and of the diaphragm.

The chapter on diseases of the lungs covers a wide variety of things, in general well and sufficiently discussed, although one might urge fuller details on pulmonary infarction, because practitioners in general do not know the clinical picture or realize its frequency. The subject of bronchopneumonia, one especially hard to handle without the inclusion of much that is uncertain or unimportant, is discussed sanely and well. Treatment is adequately covered and is notably free from nonsense. The value of the various vaccines in bronchopneumonia is dismissed briefly with the conservative statement that "their value is far from demonstrated."

Rose Bradford has a chapter on "Massive Collapse of the Lung," a subject which has been but sparsely, if at all, mentioned in American literature. It makes interesting reading, but one wonders whether the heading should not be "Massive Infarct of the Lung."

Diseases of the pleura are discussed by Capps, and as might be anticipated, more stress than the space warrants is put on the matter of referred pain, interesting as this subject is. In general, the chapter is well balanced and adequate, but it must have required self control to keep the paragraphs on the military experience with empyema down to a page and a quarter. The rather extensive discussion of atypical forms of pleurisy is interesting and

well justified because in most works on medicine these cases are not mentioned at all or only too briefly to convey any adequate conception of them.

One must feel sympathy for McLester, who has the chapter on diseases of the mediastinum, and the reviewer doubts whether any one could have done better in the twenty pages allotted. It is quite impossible to give anything more than the haziest description in this amount of space. However, the only numerically important disease of the mediastinum, aneurysm of the thoracic aorta, will probably have a chapter to itself.

I. C. Walker properly writes on bronchial asthma and hay fever, discussing them from the standpoint of protein sensitization. The chapters are readable and interesting, even if one does not fully agree that the condition is just as pictured. A useful paragraph on the determination of protein sensitivity is included.

An unusual and most valuable chapter is contributed by Ivy Mackenzie on the circulation in infections and toxic processes. It is not that it contains any new facts, but well-known facts are grouped in such a way as to stimulate thought on the part of the reader, which is far more important and far-reaching than giving information. The portions of this chapter devoted to a consideration of the relation of rheumatism, chorea and circulatory defects do not clarify the situation. As is pointed out, rheumatism is an indefinite term, used to cover a variety of clinical pictures, but probably it would have been better if the author had for this occasion made an arbitrary definition. The statements could then have been made definitely, and one would have known just what he wanted to convey.

One of the most important and perhaps the most interesting chapters in this volume is that by James Mackenzie on chronic diseases of the heart. One of the most readable of modern medical writers, he has a singular power of realizing what is what and of distinguishing between the important and the unimportant, even though the latter may at the moment be in the limelight. Any one who expects to find a careful and systematic discussion of the physical signs of cardiac disease will be disappointed, but if he is looking for information as to the essential things, such as the condition of the heart and the patient, he will find much to guide and to ponder. The important thing about a heart is what it can do, rather than just what its pathology is, and it is to this aspect of the question that Mackenzie devotes his time.

The last third of this volume is devoted to diseases of the blood. The articles are carefully written and are all just what one would expect.

A GENERAL INTRODUCTION TO PSYCHO-ANALYSIS. By PROF. SIGMUND FREUD, LL.D. Authorized translation with a preface by G. STANLEY HALL, President, Clark University. Cloth. Price, \$4.50. Pp. 402. New York: Boni & Liveright, 1920.

This volume consists of a series of twenty-eight lectures for laymen delivered in a simple, conversational way, and is conveniently divided into three parts.

Part 1 (forty-eight pages) is devoted to the "Psychology of Errors." In three lectures the author discusses those frequent and familiar phenomena which are observed in every normal person. Three classes of errors are considered: In the first are included errors of speech, errors in writing, misreading and misspelling. The second class is based on temporary forgetfulness, such as failing to think of a name which one knows and always recognizes, or forgetting to carry out a project at the proper time but remembering it later. The third class deals with erroneous ideas and permanent forgetful-

ness, such as mislaying things which cannot be found. Freud shows that errors are motivated in the unconscious and are the result of a conflict between two opposing tendencies, one of which is suppressed. The suppressed attains expression in the error.

Part 2 (142 pages) deals with the subject of dreams. He discusses the hypothesis of dream interpretation, the dream work, the dream censor, symbolism and infantilism in dreams, archaic remnants and wish fulfillment. One lecture is devoted to the analysis of sample dreams. The dream is conceived as a compromise between interfering tendencies and brings about the concealed fulfillment of a wish. Freud defines dreams as the "removal of sleep disturbing psychic stimuli by way of hallucinative satisfaction." In his lecture on dreams of childhood he concludes that childhood dreams are undistorted, unconcealed, direct wish fulfillment. The distortion of dreams is the result of the dream censor and is directed against the unacceptable of the unconscious wish impulses.

Part 3 (193 pages) is devoted to the general theory of the neuroses. In the first lecture, he discusses the relation of psychoanalysis to psychiatry. He further presents the meaning and interpretation of symptoms, their external and internal conditions and the mechanism of symptom formation. He states that psychoanalysis has been built up on the study of compulsion neuroses and hysteria. Several lectures are devoted to such subjects as resistance and suppression, transference and the development of the libido. In the lecture on traumatic fixation his aim is to show that traumatic neuroses arise from inability to meet an overpowering emotional experience. He further explains, in the lecture on the sexual life of man, that neurotic symptoms are substitutions for sexual satisfaction, normal or perverse. The theories of development and regression of the libido and of narcissism are clearly and adequately set forth. The last chapter contains a brief sketch of the work of analytic therapy. The author disparages the popular prejudiced view of psychoanalysis and hopes that the continued spread of the psychoanalytic doctrine will prove its worth. He sets forth its difficulties and its limitations. He realizes that a therapeutic novelty is received with great enthusiasm or profound distrust.

The greatest value of this volume lies in its conciseness, its clear exposition, and the comparative absence of technical terms. It cannot be considered an altogether unpartisan presentation for it takes little account of the Zurich school or of other psychologists. Although the material of the book is not new to workers in this field, it is a distinct contribution to the resources now available, and will prove caviar not only to the "ever increasing group of intelligent lay readers," but also to the average general medical practitioner.

THE SYMPATHETIC NERVOUS SYSTEM IN DISEASE (Oxford Medical Publications). By W. LANGDON BROWN, M.A., M.D. (CANTAB), F.R.C.P. (LONDON), Physician with charge of Outpatients, St. Bartholomew's Hospital, Physician to the Metropolitan Hospital. Cloth. Price, \$4.25. Pp. 161, with 9 illustrations. London: Oxford University Press, 1920.

The author pays tribute to the fundamental work on this subject by Gaskell and Langley, whose conclusions form the basis of the plan of the autonomic system as expounded in this book. He adopts Cannon's thesis that the results of sympathetic stimulation are, like the effects of epinephrin, katabolic, and serve to activate the body for a struggle and to increase its powers of defense. The action of the cranial visceral fibers, on the other hand, is anabolic and serve to build up reserves, fortifying the body against times of need and stress.

The relation of the sympathetic nervous system to the endocrine glands is discussed, and a brief summary is given of the physiology and clinical significance of the suprarenals, thyroid gland and pituitary body.

Separate chapters are devoted to the relation of the sympathetic nervous system to glycosuria, to diseases of digestion, and to the diseases of the circulatory system. It is said that sympathetic stimulation "both by increasing the secretion of glands which diminish carbohydrate tolerance and by inhibiting the gland which increases carbohydrate tolerance, would raise blood-sugar above the leak-point and glycosuria would result," and that persistent glycosuria may be of either organic or sympathetic origin. A good account is given of the innervation and movements of the alimentary canal; the part played by the sympathetic nervous system in esophageal and gastric spasm, reflex dyspepsia and hyperchlorhydria and atonic dilatation of the stomach are discussed. The vasomotor apparatus is shown not to be at fault in surgical shock.

A short chapter is devoted to vagotonia, which the author does not regard as a well established clinical entity.

The following paragraph gives a good idea of the point of view adopted and general conclusions reached in this book: "The evil effects of depressing emotions, of anxiety, fear, pain, and anger, receive an explanation when we see that through the sympathetic nervous system they can lead even to structural change. Designed as an intensive preparation for action or defense, the sympathetic response may be so dissociated, perverted, or prolonged as to produce through the thyroid gland Graves' disease with its dangers to life, through the pituitary body diabetes insipidus with its attendant discomforts, through the pancreas and other endocrine glands excessive mobilization of the blood sugar, which is the first stage of the metabolic disorder that culminates in diabetes; it may disorganize digestion by exciting spasms and atony in stomach and bowels, and inhibiting the secretion of digestive juices; it may keep blood pressure at a level which is inappropriate for the task of the heart and the arteries."

INFECTIOUS DISEASES. A PRACTICAL TEXTBOOK. By CLAUDE BUCHANAN KER, M.D., F.R.C.P., Medical Superintendent, City Hospital, Edinburgh, and Lecturer on Infectious Diseases to the University of Edinburgh, Major, R. A. M. C. T. F. Second edition. Price, \$14. Pp. 609. London: Henry Frowde, Oxford University Press, Hodder & Stoughton, Warwick Square, E. C., 1920.

In this second edition the author has made extensive alterations and additions. In addition to sections on measles, rubella, scarlet fever, smallpox, vaccinia and chickenpox, there are chapters on typhus and typhoid fever, diphtheria, erysipelas, whooping cough, mumps and cerebrospinal meningitis. In an introductory chapter, the problems of immunity are briefly but clearly discussed, as are also the symptoms and management of fever and the principles of diet and general therapy.

While special points of diagnosis in each disease are fully described, the main emphasis is continually placed on the disease picture as a whole, and the reader realizes that he is listening to the words of a finished clinician, who speaks from rich experience. Thus in discussing the diagnosis of diphtheria, the author stresses the point, well recognized but often neglected in practice, that a positive report of diphtheria bacilli does not necessarily mean that the patient is ill with diphtheria, and similarly, one negative laboratory

report does not indicate the absence of diphtheria. The findings of the laboratory must be interpreted in connection with clinical observations. The decreasing importance assigned to fomites and to house disinfection and the increased weight given to carriers in the spread of infectious disease is discussed in connection with each disease. The immunity acquired from attacks of contagious disease is interestingly discussed. In measles, while recurrences in from two to four weeks after the initial attack are noted, the author is apparently of the opinion that in general the immunity conferred by one attack of measles is permanent. While this is no doubt true in civil practice, the experience gained in concentration camps during the war would indicate that under these unusual conditions second attacks of measles are not rare.

The plates—a number of those depicting the rashes and the Schick reaction are in color—add to the teaching value of the book, and will be of special value to students. The advances in our knowledge of the etiology and transmissibility of infectious diseases are fully dealt with and throughout the book the references to American work on this and other topics show that the author is fully informed on the investigations of the best American, as well as British and other, workers.

The mechanical features of the book conform to the usual standard of Oxford publications.

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A DESCRIPTION OF SOME DISSECTIONS OF THE INTERNAL CAPSULE, THE CORONA RADIATA AND THE THALAMIC RADIATION TO THE TEMPORAL LOBE*

STEPHEN WALTER RANSON, M.D., PH.D.

CHICAGO

In the early years of the nineteenth century, dissection of the brain was practiced with great success by such men as Burdach and Reil, who were able to trace some of the larger fiber tracts in alcohol-hardened brains. But with the introduction of more refined methods, attention was diverted to the study of serial sections under the microscope, and for many years no effort was made to trace fiber bundles by mechanical dissociation. Recently there has been a revival of interest in this method of study. It has been found that in formaldehyd-hardened material bundles of fibers can be followed with ease and the outline of nuclear masses readily determined. Johnston¹ and Hoeve² advocated fiber tract dissection as a part of the laboratory course in neuro-anatomy, and in 1909 Curran,³ using this method, discovered a bundle of association fibers in the cerebrum, extending from the frontal into the occipital lobe along the inferior border of the lateral surface of the lentiform nucleus. To this tract he gave the name fasciculus occipitofrontalis inferior.

In making the dissections here described, the technic advocated by Curran has been followed. In removing nuclear masses to display fiber tracts which run through or beneath them, it is desirable to let a stream of water play over the specimen while the dissection is in progress. This serves to wash away the nuclear material as it is broken up and makes it much easier to display the fiber bundles.

The shape of the internal capsule and its relation to the basal ganglions can be studied to particular advantage in dissections prepared for this purpose. It is very difficult to form, from a study of serial sections, a clear cut mental picture of these structures in their entirety, and for

* Contribution 82, from the Anatomical Laboratory of Northwestern University Medical School, Jan. 6, 1918.

1. Johnston, J. B.: *Anat. Record* **2**:344, 1908.

2. Hoeve, H. J. H.: *Anat. Record* **3**:247, 1909.

3. Curran, E. J.: *J. Comp. Neur.* **19**:645, 1909.

this reason preparations which show their true form and relations are particularly instructive. A reconsideration of the anatomy of the internal capsule seems desirable because no articles dealing with this subject have appeared in the last twenty years, and the descriptions found in most textbooks are very unsatisfactory. A good account was given by Dejerine,⁴ but this is difficult to follow because it is based entirely on the study of sections and gives no view of the internal capsule as a whole.

DESCRIPTION OF DISSECTIONS

By removing the insula, along with the more lateral portions of the frontal, temporal and occipital lobes, one can readily expose the corona radiata, external capsule, fasciculus occipitofrontalis inferior and the fasciculus uncinatus as shown by Figure 2 in another article in this issue.⁵ The outline of the lentiform nucleus is clearly visible beneath

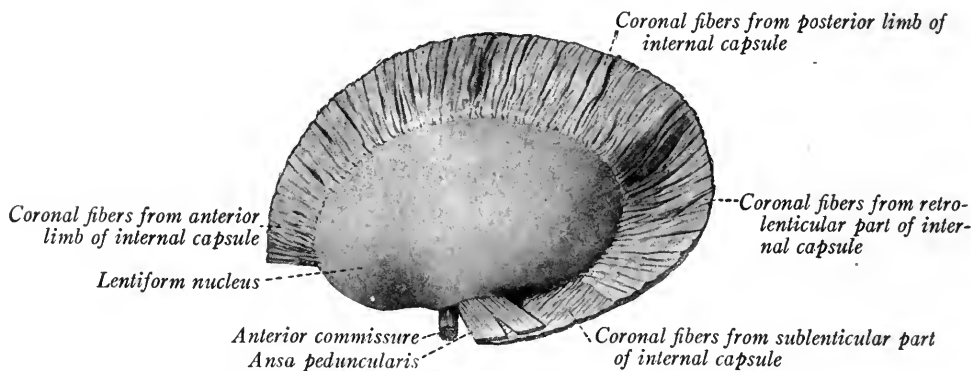


Fig. 1.—Lateral view of the lentiform nucleus and the corona radiata dissected free from the left cerebral hemisphere (from Ranson's Anatomy of the Nervous System).

the external capsule. There is a very definite plane of cleavage between the fiber bundles shown in this figure and those more superficially placed. This makes the dissection easy except in that part of the corona radiata which lies above and anterior to the lentiform nucleus. Here the fibers intersect with those from the corpus callosum and are not easily followed. The bed of fibers exposed in the dissection below and behind the lentiform nucleus is quite sharply separated from the more superficial white substance and includes both the internal and external sagittal strata of the occipital lobe.

When the uncinate and inferior occipitofrontal fasciculi have been removed and the external capsule stripped away, the lentiform nucleus is

4. Dejerine, J.: *Anatomie des Centres Nerveux* 2:19, 1901.

5. Davis, L. E.: *Anatomical Study of Inferior Longitudinal Fasciculus*, this issue, p. 370).

exposed. Its shape is well shown in Figure 1. The corona radiata is seen emerging from the internal capsule which lies medial to this nucleus. It is a broad band of radiating fibers and forms three-fourths of an ellipse surrounding the nucleus except for a short distance below and in front.

If now the lentiform nucleus is scraped away, the internal capsule will be exposed (Fig. 2). It presents an evenly concave lateral surface

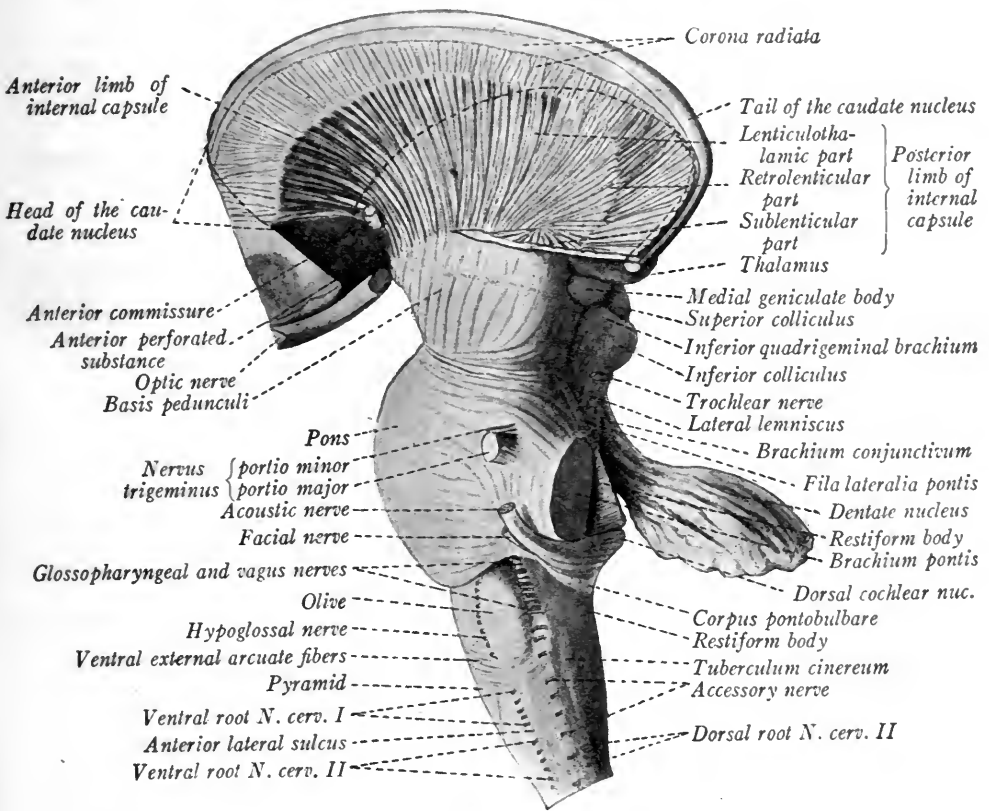


Fig. 2.—Lateral view of brain stem and internal capsule (from Ranson's Anatomy of the Nervous System).

within which the lentiform nucleus is received. There is no sharp bend in this surface to correspond to the genu, the position of which cannot be identified on this side of the internal capsule. There is, however, a rather sharp curved ridge around the border of the concave lenticular impression, and this ridge marks the transition of the internal capsule into the corona radiata. The majority of the fibers of the corona radiate forward, upward and backward in a nearly vertical plane to intersect with the radiation of the corpus callosum beyond the convex border of

the caudate nucleus; but the lowermost coronal fibers are directed lateral into the temporal lobe (Fig. 1).

The corona radiata has been partly cut away in Figure 2, so as to show the convex border of the caudate nucleus. This nucleus is for the most part hidden behind the internal capsule and the remains of the corona radiata. The outline of the hidden part is indicated by a dotted line. As this dotted line curves upward and backward over the lenticular surface of the internal capsule, it marks the position of the genu which appears as a curved ridge when the capsule is viewed from the medial surface (Fig. 3). Between this dotted line and the curved ridge marking the edge of the lenticular impression is the lenticulocaudate portion or anterior limb of the internal capsule (Fig. 2). Behind the

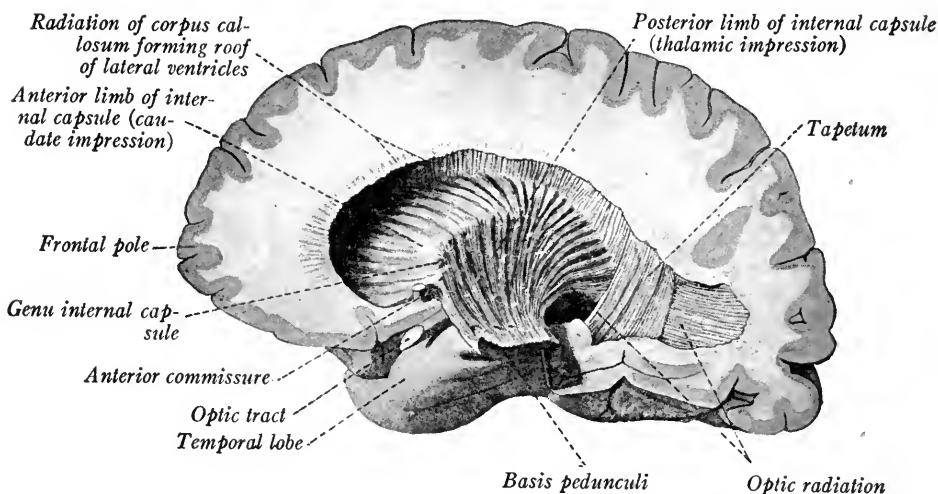


Fig. 3.—Dissection of cerebral hemisphere, showing the internal capsule exposed from the medial side. The caudate nucleus and thalamus have been removed (from Ranson's Anatomy of the Nervous System).

dotted line is the posterior limb of the internal capsule which has been subdivided by Déjerine⁴ into lenticulothalamic, retrolenticular and sublenticular portions. The figure shows very well the direct continuity of the internal capsule with the basis pedunculi on the one hand and the corona radiata on the other.

An instructive view of the internal capsule can be obtained from the medial side by opening up the lateral ventricle and scraping away the thalamus and caudate nucleus. Each of these nuclear masses makes a distinct impression on the medial surface of the capsule, and between the two is a prominent curved ridge (Fig. 3). In horizontal sections through the brain at appropriate levels, this ridge appears as a sharp angle between the anterior and posterior limbs of the internal capsule

and is known as the genu. This genual ridge follows the line of the sulcus which separates the thalamus from the caudate nucleus in the floor of the lateral ventricle and which contains the stria terminalis. Figure 3 shows why the shape of the internal capsule and the position and prominence of the genu vary as they do in horizontal sections taken at different levels through the basal ganglions.

The caudate impression corresponds in outline to the shape of the nucleus which produces it. Where the head of the nucleus rests on the capsule, the impression is large, and as the nucleus tapers to form the long curved tail, the caudate impression also becomes narrow.

The thalamic impression is markedly concave and belongs entirely to the posterior limb of the capsule. Below, it goes over without sharp line of demarcation into the posteromedial surface of the basis pedunculi at about the level of the optic tract. Above, it is limited by the curved ridge of the genu. The width of this portion of the capsule increases steadily from below upward. Its anterior border is continuous with the anteromedial border of the basis pedunculi. At its upper extremity a short distance behind the anterior commissure, this border bends forward at a rather sharp angle to become continuous with the inferior border of the anterior limb. The entire thalamic surface of the internal capsule as seen after removal of the thalamus and subthalamus is shaggy in appearance, due to the presence of innumerable fibers of the thalamic radiation which were broken off when the thalamus was removed.

The anterior limb, *pars frontalis*, or lenticulocaudate portion of the internal capsule, consists in part of fibers from the medial portion of the basis pedunculi (frontopontine tract) which curve forward over the anterior commissure and run a more or less horizontal course toward the frontal lobe. But the majority of the fibers belong to the frontal stalk of the thalamus which is also directed horizontally forward. This segment of the internal capsule has a sickle-shaped outline and presents a distinct lower border beneath which the caudate and lentiform nuclei are fused together (Fig. 2). Its other two borders are curved with convexity forward and upward. Of these the anterosuperior border presents the greater curvature and corresponds to the anterior part of the ridge between the internal capsule and corona radiata. The posterior inferior border is indicated in Figure 2 by a dotted line which corresponds to the position of the genual ridge. These two borders meet in an acute angle which points toward occiput.

Horizontal sections through the internal capsule above the level of the anterior commissure cut through the thalamus as well as the genu and both limbs of the internal capsule. Such sections pass through what Déjerine⁴ has called the thalamic region of the internal capsule.

Sections through the anterior commissure or immediately below it pass through both the thalamus and subthalamus, since the latter is situated in front of, as well as below, the former. Such sections are said to pass through the subthalamic region of the internal capsule. They do not include either the genu or the anterior limb.

The posterior limb of the internal capsule is more complex than the anterior and, as has already been said, consists of three parts: lenticulothalamic, retrolenticular and sublenticular. The lenticulothalamic portion includes that part of the lenticular surface which lies behind the curved line of the genual ridge (Fig. 2). Its fibers have for the most part a vertical direction. They belong to the corticobulbar, corticospinal, and corticorubral tracts and to the parietal stalk of the thalamus; and to these there are added in the subthalamic region the frontopontine tract from the anterior segment and the temporopontine fibers from the sublenticular segment. It is usually said that the fibers of the corticobulbar tract pass through the genu of the internal capsule. But since the genu is a curved ridge of considerable extent, it will be obvious that this statement can apply only to the genu as it is seen in horizontal sections through a particular region. According to Déjerine the corticobulbar tract passes through the genu in the inferior one-third of the thalamic region.

The retrolenticular segment of the internal capsule lies behind the lentiform nucleus on the lateral surface of the thalamus. It consists of fibers from the pulvinar, which are at first directed horizontally outward behind the lentiform nucleus and then turn sharply backward to run over the lateral surface of the tail of the caudate nucleus toward the occipital lobe. They form the optic radiation.

The sublenticular part of the internal capsule extends laterad beneath the lentiform nucleus (Figs. 1 and 2), and helps to form the roof of the inferior horn of the lateral ventricle. It is composed of fibers which enter it from the lateral part of the basis pedunculi (the temporopontine tract) and of fibers uniting the thalamus with the temporal lobe. The latter group of fibers had been dissected away in the preparation from which Figure 2 was drawn, and only the temporopontine fibers are illustrated. They spread out on the upper surface of the sublenticular segment immediately beneath the lentiform nucleus and are directed nearly horizontally laterad into the temporal lobe. Traced in the other direction in the dissected specimen, they can be seen to turn downward and backward into the lateral part of the basis pedunculi.

Beneath this layer of temporopontine fibers is another composed of fibers joining the thalamus with the temporal lobe. These fibers sweep forward and laterad under cover of the temporopontine tract which has been removed in Figure 1, in order to display them. There are

thus two distinct strata in the sublenticular segment, the upper of which is composed of laterally directed temporopontine fibers while the lower belongs to the thalamic radiation to the temporal lobe and has its fibers directed forward and laterad.

In this article, we are especially concerned with those portions of the thalamic radiation which go to the temporal lobe. The ventral stalk or inferior peduncle of the thalamus, also known as the *ansa peduncularis* (Fig. 1) springs from the basal aspect of the anterior part of the thalamus and runs behind the anterior commissure under the posterior end of the anterior limb of the internal capsule and then laterad beneath the lentiform nucleus to the insula and temporal lobe. In addition to this well known bundle, there are other fibers connecting the thalamus with the temporal lobe. Degenerations resulting from experimental lesions in the brains of monkeys have shown, according to Probst,⁶ that thalamotemporal fibers run through the posterior part of the internal capsule to the cortex of the superior temporal gyrus. Some of these fibers are said by him to leave the internal capsule above the lentiform nucleus and run through the external and extreme capsules to the superior temporal gyrus.

But by far the largest bundle of fibers joining the thalamus with the temporal lobe is the temporothalamic fasciculus of Arnold,⁷ which we have already encountered in the sublenticular segment of the internal capsule (Fig. 1). This bundle emerges from the ventrolateral part of the posterior extremity of the thalamus under cover of the external geniculate body. The fibers form a large strand directed forward in the inferior of the two layers of the sublenticular segment of the internal capsule along the roof of the inferior horn of the lateral ventricle. Here a few at a time they curve outward and then somewhat backward into the white matter of the temporal lobe. Thus as they spread out toward their distribution, the fibers describe broad curves with convexity forward. The most medial of these fibers extend farthest forward reaching the anterior extremity of the roof of the inferior horn before they curve outward and backward toward their distribution (Fig. 4). The fasciculus of Arnold must not be confused with the fibers from the medial geniculate body, which constitute the auditory radiation and which are mingled with the temporopontine tract in the sublenticular segment of the internal capsule.

Another bundle of thalamotemporal fibers seems to have been overlooked by nearly all previous investigators. The constituent fibers of this fascicula can be traced from the stratum zonale on the dorsal sur-

6. Probst, M.: Sitzungsab. d. k. Akad. d. Wissensch. Math.-natur. w. Wien. **114**:173, 1905.

7. Arnold, A.: Handbuch der Anatomie des Menschen **2**: 1851.

face of the posterior part of the thalamus. They are gathered into a compact bundle along the medial side of the stria terminalis. The bundle arches backward, then downward, and finally forward around the posterior extremity of the thalamus following the tail of the caudate nucleus into the roof of the inferior horn of the lateral ventricle (Fig. 4). In its arched course around the thalamus it lies on the medial side and partly under cover of the stria terminalis. Passing through the sublenticular portion of the internal capsule, the bundle flattens out in the roof of the inferior horn of the lateral ventricle under cover of the ependymal lining and can be traced forward to the anterior part of the temporal lobe. It lies on the ventricular surface of the temporothalamic fasciculus of Arnold between that bundle and the epen-

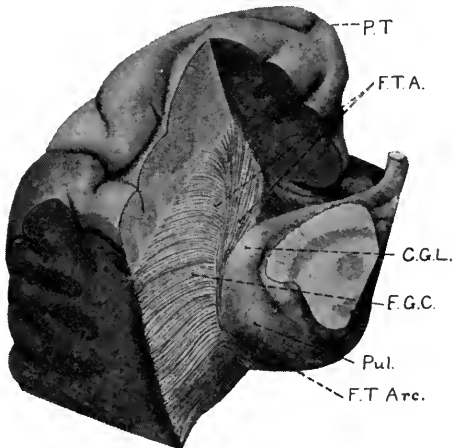


Fig. 4.—Dissection of the temporal lobe from below. The mesencephalon has been cut across at the level of the superior colliculus. The medial and inferior portion of the temporal lobe has been removed and the fibers of the corona radiata and sublenticular part of the internal capsule exposed: *C. G. L.*, corpus geniculatum laterale; *F. T. A.*, fasciculus temporothalamicus of Arnold and fasciculus thalamotemporalis arcuatus; *F. T. Arc.*, fasciculus thalamotemporalis arcuatus; *F. G. C.*, fasciculus geniculocalcarinus; *P. T.*, polus temporalis and *Pul.*, pulvinar.

dyma of the ventricle. In order to differentiate this bundle from that described by Arnold, it may be designated as the fasciculus thalamotemporalis arcuatus.

After these fibers from the stratum zonale had come to view in our dissections, a careful review of the literature was made to see if they had been described before. The only mention of them seems to be by Probst⁶ in 1905. He traced them in the brain of a monkey after an

experimental lesion in the thalamus and internal capsule and regarded them as constituting an hitherto undescribed bundle which he designated in his drawings by the letter y. His description of this bundle corresponds exactly with that given herewith for the fasciculus thalamotemporalis arcuatus. The degeneration which he found in this fascicle resulting from a lesion in the thalamus indicates that at least a large part of these fibres are corticopetal. Probst states that he has also been able to demonstrate these fibers in the brains of microcephalic idiots where for some reason they stand out with special clearness.

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AN ANATOMIC STUDY OF THE INFERIOR LONGITUDINAL FASCICULUS *

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In the majority of anatomic textbooks the inferior longitudinal fasciculus is described as one of the cerebral association tracts. Its course is spoken of as extending between the poles of the occipital and temporal lobes. The exact anatomic relations of this bundle and the possibility that it represents projection fibers joining the thalamus with the cortex of the occipital and temporal lobes have caused a great difference of opinion among anatomists and neurologists. All observers to date are agreed that it consists of a well defined aggregation of nerve fibers. With two exceptions, all of the recent work on this subject has been done by examinations of serial sections, obtained from cases of cerebral softening or tumors, showing degenerative changes. It should be comparatively easy, however, to show by gross dissections of well hardened specimens, a bundle such as the fasciculus longitudinalis inferior.

In this work we have undertaken to settle by gross dissections of the cerebrum, these moot points with reference to this particular fasciculus: first, the course, extent and relations of the inferior longitudinal bundle, and second, whether or not this tract consists of projection or association fibers. Naturally, we assume in the beginning that such a bundle really exists as a distinct entity.

Reil¹ (1809) was the first to describe the inferior longitudinal bundle. He did not recognize it as such, but included it in his description of the corona radiata of the occipital lobe.

Burdach² (1822) identified the fasciculus longitudinalis inferior as a definite bundle and termed it an association tract. He describes it thus:

In each hemisphere, it extends along the base of the corona radiata; the lower longitudinal fibers coursing in uninterrupted fashion from the pole of the occipital lobe. It forms, in the longitudinal direction, a prominent eminence upon the under surface of the cerebrum. It is somewhat curved longitudinally, arched externally and slightly concave laterally. It forms in its arch, a very level curve which corresponds to the external capsule and is contrasted

* Contribution 83, from the Anatomical Laboratory of the Northwestern University Medical School, Jan. 6, 1921.

1. Reil: *Archiv für die Physiologie* von Prof. J. C. Reil und Dr. J. H. F. Antenreith (Halle), quoted from Niessl-Mayendorf (Footnote 7).

2. Burdach: *Von Bau und Leiten des Gehirns* 1:152, 1822, quoted from Niessl-Mayendorf (Footnote 7).

to the fasciculus uncinatus which is somewhat concave inferiorly and slightly convex superiorly. It begins in the pole of the occipital lobe and runs anteriorly along the external part of the floor of the inferior horn. In the temporal lobe, it courses somewhat externally and becomes the foundation of the outer wall or the external division of its floor, and bears the hippocampus. It forms a groove in which the corona radiata progresses. Its inner part forms the inner margin of this groove and unites with the tapetum and cingulum. Its outer part unites with the ground bundles which come up in the lateral border of the temporal lobe. A portion of the fibers run more obliquely, anteriorly and medially beneath the fasciculus uncinatus; and course onward beneath the lenticular nucleus to reach the island of Reil. They form the floor of the external capsule and curve somewhat externally and enter the frontal lobe where they course above the uncinate fasciculus and extend to the external side of the frontal pole.

It will be evident from Figure 2, which illustrates the dissection of the lateral aspect of the cerebrum, that Burdach has very accurately described the fasciculus occipitofrontalis inferior.

Gratiolet³ (1839) says nothing of a long association bundle from the occipital to the temporal lobe. He describes, however, two divisions of the optic radiation, whose fibers intermingle in the occipital lobe and run forward to terminate within the external geniculate body, the pulvinar and the anterior corpora quadrigemina.

Sachs⁴ (1892) believes the inferior longitudinal bundle to be a fasciculus of association fibers originating with the occipital lobe and terminating within the temporal lobe, especially in the first and second temporal convolutions. The inner portion of the tract is in close relation to the cingulum, while the fibers of the superior portion intermingle with those of the corona radiata. Sachs introduced the term "external sagittal stratum" in describing the superior fibers.

Dejerine⁵ (1895) describes the bundle as a fasciculus of association fibers arising within the pole and entire cortex of the occipital lobe. Within the temporal lobe and at the level of the retrolenticular segment of the internal capsule, the inferior longitudinal bundle curves around the posterior extremity and the inferior border of the putamen, surrounds the sphenoidal horn and reaches the external part of the amygdaloid nucleus where with the fasciculus uncinatus they divide the gray substance of the neighboring cerebral cortex. Within the temporal lobe, the fibers of the inferior couch course within the hippocampal convolution, the fusiform lobe and the third temporal convolutions. A

3. Gratiolet: *Anatomie comparée du système nerveux*, Paris, 1839, quoted from Niessl-Mayendorf (Footnote 7).

4. Sachs, H.: *Ueber Flechsig's Verstandescentren*, *Monatschr. f. Psychiat. u. Neurol.* 1:199, 1892.

5. Dejerine, J.: *Anatomie des centres nerveux*, quoted from Archambault (Footnote 11).

large number of fibers radiate within the first and second temporal convolutions. A small number of fibers enter into the constitution of the external capsule and intercross anteriorly with the fibers of the anterior commissure and the fasciculus uncinatus.

Flechsig⁶ (1896) working on the myelinization of fibers, wrote about the bundle under the term "primary optic radiation." He described the fibers as originating within the inferior part of the external geniculate body and the principal nucleus of the pulvinar and terminating within the visual sphere of the occipital lobe. In their course, they cross fibers originating from the superior part of the external geniculate body which enter into the stratum zonale. They are joined by fibers coursing from the internal geniculate body to the temporal lobe, hippocampus and the olfactory area. The "secondary optic radiation," or the optic radiation proper of Gratiolet, was described by him as originating within the occipital lobe and coursing to the pulvinar of the thalamus. The fibers of the primary optic radiation receive their myelin sheaths very early and before those of the secondary radiation. Flechsig believes that the primary fibers carry the impulses from the macula lutea to the visual area. Their clinical importance is therefore obvious.

Niessl-Mayendorf⁷ (1903) continued Flechsig's work and more accurately described the fasciculus longitudinalis inferior, which he maintained was identical with Flechsig's "primary optic radiation." He also stated that the cells of origin were situated anteriorly. This hypothesis was supported by the appearance of degeneration and atrophy found in the fibers of this tract following lesions of the external geniculate body. Mayendorf believes that the inferior longitudinal bundle consists, therefore, of projection fibers arising within the external geniculate body and the thalamus and terminating in the calcarine fissure of the occipital lobe. He refers to the "secondary optic radiation" of Flechsig, which lies more medially, as the optic radiation proper. These fibers he described as centripetal in direction, arising in the visual sphere and terminating within the thalamus and medial surface of the anterior corpora quadrigemina.

Edinger⁸ (1904) believes that the bundle in question is a long association tract uniting the temporal and occipital lobes. His conclusions were drawn from a case of ablation of the temporal cortex and from examination of the course of degenerated fibers.

6. Flechsig: Weitere Mittheilungen über den Stabkranz des Menschlichen Grosshirns, *Neurol. Centralbl.* **15**:2, 1896.

7. Niessl-Mayendorf: Vom Fasciculus longitudinalis inferior, *Arch. f. Psychiat.* **37**:537, 1903.

8. Edinger: Bau der nervösen centrale Organe, *Arch. f. klin. Med.* **73**:243, 1904.

Probst ⁹ (1902) has made a thorough study of the visual fibers and their connections. In his latest publications dealing with this subject, he reports a case which he studied, showing a lesion of the optic thalamus. He observed a partial degeneration of the inferior longitudinal fasciculus which extended into the temporal and occipital lobes. In the midst of these degenerated fibers, he found undegenerated fibers originating from the pulvinar and external geniculate body. The latter areas were not involved in the lesion. He also found undegenerated fibers originating within the cortex of the temporal and occipital convolutions which coursed to the cerebral peduncle. For Probst, therefore, the inferior longitudinal fasciculus consists of thalamocortical fibers directed toward the temporal and occipital lobes, and also of fibers originating within these lobes and passing to the cerebral peduncle. Under the optic radiation of Gratiolet, he includes the corticifugal fibers to the pulvinar and the anterior quadrigeminal body.

Redlich ¹⁰ (1905) describes the fasciculus longitudinalis inferior as a part of the optic radiation terminating within the calcarine fissure, the occipital pole, the inferior convolution of the occipital lobe and partly within the cortex of the parieto-occipital convexity. Within the temporal lobe, the bundle gives off fibers constituting the internal sagittal stratum. The dorsal portion of the fibers are in close relation with the thalamic radiation proper and from a part of the corona radiata. They enter the relation with the optic thalamus and with the external geniculate body, but Redlich believes that they are corticifugal fibers.

Archambault ¹¹ (1905, 1906, 1909) has studied the inferior longitudinal bundle most thoroughly. His material was obtained from cases of cerebral softening, and his conclusions are drawn from serial sections of these brains. This author believes that the inferior longitudinal fasciculus consists of: (1) a bundle of projection fibers, which he has termed the "fasciculus geniculocalcarinus" or the "fasciculus opticus centralis" and (2) of some unnamed fibers of association. He describes the fasciculus geniculocalcarinus as a definite bundle of fibers which

9. Probst: Ueber den Verlauf der centrale Sehfaser (Rinden-Sehhügel-fasern) und deren Endigung um Zwischen und Mittelhirne und über die Associations und commissurenfasern der Sehsphäre, *Arch. f. Psychiat.* **35**:22, 1902.

10. Redlich: Zur vergleichenden Anatomie der Associationsysteme des Gehirns der Säugethiere, *Fasciculus longitudinalis inferior*, *Arb. a.d. Neurol. Inst.* (Obersteiner) **12**: 1905.

11. Archambault, L.: Le faisceau longitudinal inférieur et le faisceau optique central, *Rev. neurol.* **13**:1053, 1905; Le faisceau longitudinal inférieur et le faisceau optique central, *Nou. iconog. de la Salpêtrière* **2**:215, 1906; The Inferior Longitudinal Bundle and the Geniculocalcarine Fasciculus: Contribution to the Anatomy of the Tract Systems of the Cerebral Hemispheres, *Albany M. Ann.* **30**:118, 1909.

occupies a portion of the external and internal sagittal beds in the temporal lobe, and which constitutes the entirety of the external sagittal stratum in the occipital lobe. This fasciculus, which represents the corticopetal corona radiata to the occipital lobe, originates within the external geniculate body and terminates within the two lips of the calcarine fissure, but especially within the inferior lip. His description of the fasciculus is as follows:

The fasciculus geniculocalcarinus takes its origin within the superior and external part of the external geniculate body. The fibers arising from the anterior part of the nucleus pass obliquely backward and inferiorly, along the external wall of the anterior extremity of the sphenoidal horn where they are joined with the fibers of the fasciculus uncinatus, the fibers of the anterior commissure and the short fibers of association of that region. They then bend slightly inward and run backward to the external part of the wall of the inferior horn of the lateral ventricle. The fibers which take their origin in the middle part of the geniculate body, enter into the constitution of the inferior part of the posterior segment of the internal capsule. Traversing the third segment of the lenticular nucleus, they turn at the level of the posterior and inferior part of the external capsule and are directed obliquely inferiorly and medially, where a part of them pass to the external sagittal couch and a part to the internal sagittal couch. Other of the fibers intermingle at this level with the fibers of Türck and with the corticopetal and corticofugal fibers of the first temporal convolution. The fibers which arise in the posterior part of the geniculate body curve around the triangular area of Wernicke and reach the retrolenticular segment of the internal capsule. They project interiorly and reunite in a compact fasciculus which embraces the inferior part and the external border of the sphenoidal caudate nucleus. The fibers are extremely dissociated by the thalamic radiation of the occipital, parietal and temporal regions, which at this level pass into the pulvinar of the optic tract.

Meyer¹² (1907) reports data derived from cases of cerebral softening, adding to Flechsig's original idea that the external sagittal layer consists of projection fibers coursing from the thalamus to the calcarine cortex. Meyer accepts Archambault's term, "fasciculus geniculocalcarinus" as designating the occipital portion of the external sagittal layer.

Curran¹³ (1909) describes a well marked bundle of fibers extending from the frontal to the occipital lobe. He has termed these fibers the "fasciculus occipitofrontalis inferior." It begins as a fan-shaped structure in the occipital lobe and becomes a well defined bundle, swinging to the lower, external side of the lenticular nucleus and the external capsule. It again spreads out fanlike in the frontal lobe. Inferiorly to this bundle lies the middle and posterior portions of the fasciculus

12. Meyer: Two Cases of Cerebral Softening. *Tr. A. Am. Phys.* **2**:2, 1907.

13. Curran: A New Association Fiber Tract in the Cerebrum, *J. Comp. Neur. and Psych.* **19**:645, 1909.

uncinatus. Anteriorly, the floor of the fasciculus occipitofrontalis inferior is formed by the corona radiata and the anterior commissural fibers. In the medial portion of its extent, the floor is formed by the descending horn of the lateral ventricle, the corona radiata, the tapetum and the tail of the caudate nucleus. Posteriorly, the fasciculus lies on the ependyma of the posterior horn of the lateral ventricle. Curran believes the inferior longitudinal bundle lies more externally and inferiorly, and that some of the fibers intercross far back in the occipital lobe with the fasciculus occipitofrontalis inferior. No illustrations of the inferior longitudinal bundle are given. This author's work was done on gross dissections of the cerebral hemisphere from the lateral aspect.

Trolard¹⁴ (1906), however, had previously made a similar gross dissection from the lateral aspect of the brain. His illustration shows in detail the fasciculus described by Curran. Trolard believes that the inferior longitudinal fasciculus, as described by Déjerine, formed a part of this bundle. The article, it seems, was only preparatory to further work; but a very careful search of the literature has not revealed the later studies.

A review of the literature, therefore, reveals opinion to be divided about equally as to whether or not the inferior longitudinal fasciculus is an association or a projection tract. Burdach, Sachs, Déjerine, Edinger, Curran and Trolard believe the bundle to be purely an association tract. On the other hand, Flechsig, Probst, Niessl-Mayerdorf, Dedlich and Archambault insist strongly that it is a bundle of projection fibers. With the exception of Curran and Trolard, whose articles include illustrations of gross dissection, the more recent authors have used degenerated material and have based their conclusions on serial sections. Curran emphasizes the fact that well defined fiber tracts in the cerebrum can be clearly defined in gross dissections of well hardened specimens.

TECHNIC

In this work, we have made several gross dissections of human brains in an effort to attain our objectives. The results of our dissections in each case have been identical. The final dissections, from which the accompanying illustrations were made, are, therefore, entirely representative. These dissections were made on adult brains, which were removed from one to four hours following death. The fresh specimens were immediately suspended in a large jar containing 10 per cent. formaldehyd solution, by attaching a cord to the vessels comprising the circle of Willis. The fluid completely surrounded the brain, and no part of it was allowed to touch any part of the container. This point of

14. Trolard: Le faisceau longitudinal inférieur du cerveau, *Rev. neurol.* **14**:440, 1906.

technic was insisted on to prevent distortion of the brain and to preserve the exact relations of all fibers within the cerebrum. The specimens were allowed to remain suspended in this manner for five weeks. The fluid was changed twice during this period.

Dissections of the fiber tracts were made in accordance with the detailed technic described by Curran. After the dissections were made, photographs were taken of the brain and the drawings were made from the picture and the specimen. The object of this procedure was to get the exact structural proportions and relations which are difficult to obtain from a drawing made directly from the specimen.

In our dissections, it clearly became our first duty to identify those fibers which have been included in previous descriptions of the inferior longitudinal fasciculus. By a careful evaluation of the literature heretofore reviewed, one will see that three descriptions of this fasciculus have been given.

First, Archambault's description, in which he gives in detail the relations of the fasciculus geniculocalcarinus. This, he believes to be the principal portion of what has been considered the inferior longitudinal bundle by many authors. This tract agrees with Flechsig's primary optic radiation and with Niessl-Mayendorf's description of the inferior longitudinal fasciculus.

Second, a long association tract of fibers coursing between the occipital and temporal lobes described by Déjerine, Sachs and their followers. I shall show later that at least the majority of these fibers extend anteriorly into the frontal lobe and do not end within the temporal lobe.

Third, Curran's description of the fasciculus occipitofrontalis interior, earlier seen by Trolard and apparently also by Burdach, in which he states that the inferior longitudinal fibers are placed more inferiorly than the main bundle and consist of shorter occipitotemporal association fibers.

DESCRIPTION OF SPECIMENS

Figure 1 is a drawing made from the medial aspect of the left cerebral hemisphere. The splenium of the corpus callosum, the fimbria, the inferior and posterior horns of the lateral ventricle, the cingulum and the hippocampus have been removed, as well as the overlying cortex in this region. The stria terminalis, the temporothalamic fasciculus of Arnold, and the arcuate thalamotemporal fasciculus described by Ranson have also been removed. We can very easily identify the thalamus, pulvinar, lateral geniculate body, cerebral peduncle, the optic nerve and the anterior commissure.

In the occipital lobe, the thalamic radiation to the visual area is clearly seen, extending from the cortex to the thalamus in its posterior

and superior surface. These fibers constitute the optic radiation proper, as described by Gratiolet, and have been determined to be corticifugal in direction. As we pass inferiorly and externally, we encounter a definite layer of fibers in the same plane as these fibers. This bundle runs from the lateral lip of the inferior and posterior surfaces of the thalamus and from the lateral geniculate body to the visual area. These fibers, very characteristically, curve forward and inferiorly before they sweep back to their termination. We may definitely identify them as the fasciculus geniculocalcarinus, whose cells of origin according to Archambault, rest within the external geniculate body.

Farther inferiorly and somewhat externally, we see long uninterrupted fibers lying on the same plane as the two previously described

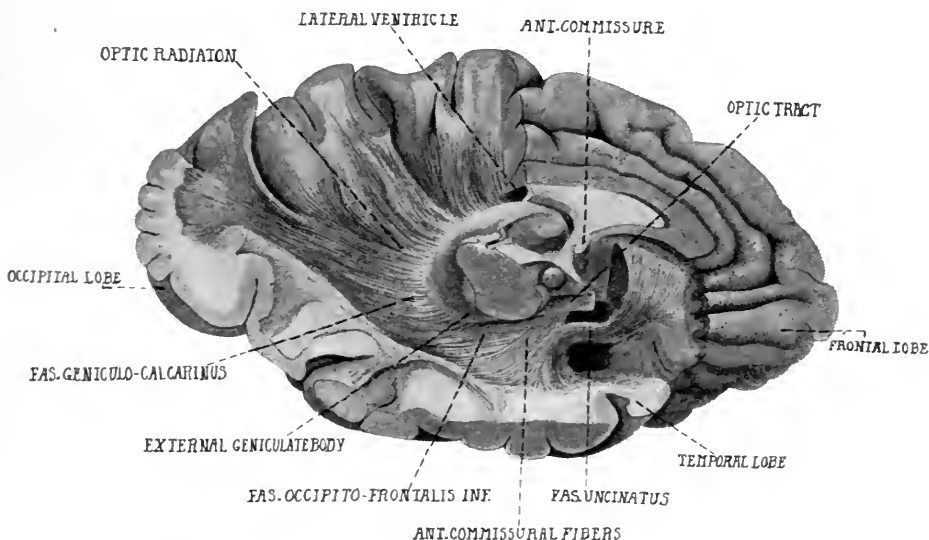


Fig. 1.—Medial view of a dissection of a human cerebral hemisphere. The splenium of the corpus callosum, the fimbria, the inferior and posterior horns of the lateral ventricle, the cingulum, the hippocampus and the overlying cortex have been removed. Reduced one-fourth.

fiber tracts. This group of fibers is in close relation above with the fasciculus geniculocalcarinus as far forward as the anterior surface of the lateral geniculate body. At this point, they continue still farther anteriorly to pass beneath the anterior commissural fibers. No fibers can be discerned to pass into the thalamus at this point. They spread out in a fan-shaped manner deep within the cortex of the frontal lobe. As these fibers course into the frontal lobe, they lie somewhat superior and anterior to the fasciculus uncinatus.

Still more inferiorly, we encounter shorter fibers wholly within the temporal lobe. These are shown very distinctly in the illustration and

must be considered only as longer arcuate fibers of association within the temporal lobe. The latter fibers are evidently the ones which Curran has referred to as being external and superficial to the longer fibers of his fasciculus occipitofrontalis inferior and which he groups under the inferior longitudinal bundle. Their evident intratemporal course, however, shows that they cannot properly be so designated.

The longer fibers just described as coursing between the occipital and frontal lobes and as bearing such close relation to the fasciculus geniculocalcarinus are no doubt the association tract which Déjerine, Edinger, Sachs and others have described as the inferior longitudinal bundle. We believe, however, that these fibers are merely the internal layer of the fasciculus occipitofrontalis inferior as described by Curran. By carefully dissecting away individual fibers of this layer, one invariably reaches the external fibers of Curran's bundle. The individual

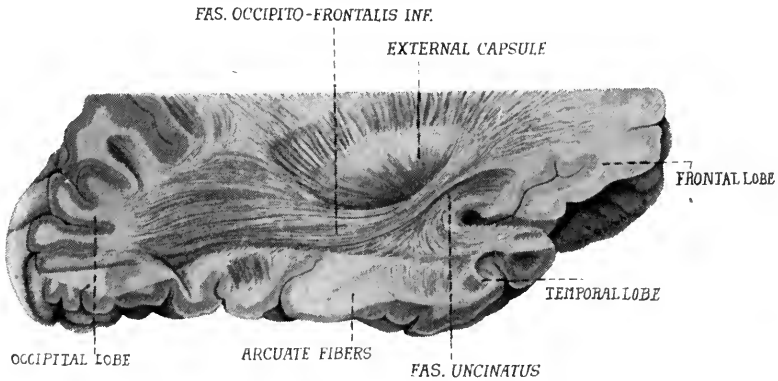


Fig. 2.—Lateral view of a dissection of a human cerebral hemisphere. The insula, opercula and adjacent parts have been removed. The dorsal portion of the hemisphere has been cut away. Reduced one-fourth.

fibers can be carried in an uninterrupted manner between the frontal and occipital lobes.

Figure 2 is the reproduction of a drawing made from a dissection of the lateral aspect of the cerebral hemisphere. The opercula, insula, superior longitudinal bundle and the fasciculus transversus occipitalis have been removed.

We see the fasciculus occipitofrontalis inferior extending between the frontal and occipital lobes. Fibers converge from a large part of the frontal lobe and swing inferiorly to the lenticular nucleus as a compact bundle. They then spread out in a fan-shaped manner into the occipital lobe. At the posterior inferior edge of the lenticular nucleus, which we see covered by the external capsular fibers, a marked depression is evident. This depression is formed by two prominences; one, situated above the depression and curving from above downward and

backward, and the other, situated below the depression and proceeding anteriorly into the temporal lobe. Still a third prominence is related to the above two and extends into the occipital lobe. These eminences are formed by the lateral ventricle with its posterior and inferior horns. Internally, the optic radiation proper and the fasciculus geniculocalcarinus occupy the concave surfaces of the superior and posterior prominences; while the inferior portion of the fasciculus geniculocalcarinus and the internal layer of the fasciculus occipitofrontalis inferior are closely applied to the inferior prominence and to the inferior portion of the posterior prominence. Externally, the fasciculus occipitofrontalis inferior sweeps over the posterior and inferior and over the inferior portion of the superior prominences.

The individual fibers of the fasciculus occipitofrontalis inferior can easily be carried forward from the occipital lobe to the frontal lobe in unbroken fashion, thus demonstrating their continuity. In its medial portion, the fasciculus uncinatus lies inferiorly and in close apposition. By carefully dissecting the fasciculus occipitofrontalis inferior away, we find, as we go deeper, that we are removing fibers which are closely related to the fasciculus geniculocalcarinus and which are shown in Figure 1 as long occipitofrontal fibers. It seems reasonable to believe that these fibers and the fibers of the fasciculus occipitofrontalis inferior are constituents of the same bundle.

Inferior to the fibers of the fasciculus occipitofrontalis inferior, within the temporal lobe, only short arcuate intratemporal fibers are found. No occipitotemporal fibers can be found lying inferiorly, such as Curran designates under the name fasciculus longitudinalis inferior.

Throughout the literature, one encounters the terms, "internal sagittal stratum" and "external sagittal stratum." The word "stratum" is used synonymously with "bundle" or "fasciculus" by many authors. Sachs, who introduced the terms, refers to the inferior longitudinal bundle as the "external sagittal stratum." The use of these loose terms has caused misinterpretations and false conclusions which are obvious. I believe the term "stratum" should define a bed or groundwork of fibers wholly distinct from a compact aggregation of nerve fibers which form a fasciculus or bundle. I believe the external sagittal stratum consists of several components which are from the occipital lobe forward; the fasciculus geniculocalcarinus, the fasciculus occipitofrontalis inferior; the corona radiata to the parietal lobe, some fibers of the geniculocalcarine fasciculus in the temporal lobe plus the corticifugal fibers of projection of the first and second temporal convolutions, and most anteriorly, the fasciculus occipitofrontalis inferior. The internal sagittal stratum, from the occipital lobe anteriorly, consists of the optic radiation proper, some fibers of the fasciculus geniculocalcarinus and

the fasciculus of Türeck. The various constituents of these arbitrary terms can easily be identified in the accompanying illustrations. It also becomes evident that the internal sagittal stratum of the occipital lobe is represented wholly by the optic radiation proper, while the external sagittal stratum in the same lobe is formed entirely by the fasciculus geniculocalcarinus.

It is clearly seen how much confusion exists as to the exact anatomy of the inferior longitudinal fasciculus. At least three distinct anatomic entities have been described under the name of this tract. The fasciculus geniculocalcarinus or the primary optic radiation of Flechsig has been definitely isolated both in degeneration sections and by our gross dissections. It is a thalamocortical projection tract, which anatomically is very hard to differentiate from the optic radiation proper. By serial sections, showing degeneration of the fibers, this becomes relatively easy. We can see no real reason for designating this tract the inferior longitudinal bundle. On the other hand, it seems logical to retain such a definite and accurately descriptive term as Archambault has applied to it.

Our dissections further show that the long fibers lying external and inferior to the fasciculus geniculocalcarinus extend between the occipital and frontal lobes. These fibers belong to the tract described from the lateral aspect by Curran, as the fasciculus occipitofrontalis inferior. The classical description of the inferior longitudinal bundle, accepted and pictured by numerous anatomic textbooks, is that of a tract extending from the occipital to the temporal poles. But the fibers in question can be definitely shown to continue in unbroken fashion from the occipital to the frontal lobes, whether examined from the medial or the lateral aspect. It has also been shown, from both aspects of the cerebrum, that no long association fibers lie inferior to this bundle either in the occipital or the temporal lobes. It seems, consequently, that those fibers are not to be named as the inferior longitudinal bundle, provided the classical description of the latter is retained. In fact, no bundle of fibers can be found by dissection, which fits the description accepted by textbook authors. This, of course, does not exclude the possibility that some fibers which join the temporal and occipital cortex may be mingled with the two long tracts just described, but these, if present, are not sufficiently numerous to make possible their recognition by the dissection method.

If we are to use accurate, descriptive, anatomic terms, we must either speak of Curran's fasciculus occipitofrontalis inferior as the inferior longitudinal bundle or we must drop the latter term, as it refers to no anatomic entity. I believe that the more definite name, fasciculus occipitofrontalis inferior, should be used to designate this

bundle, which evidently must, therefore, be purely an association tract. This is substantiated by the definiteness with which these fibers are separated from those of the fasciculus geniculocalcarinus which arise from the external geniculate body.

CONCLUSIONS

I believe, therefore, that:

1. Such purely occipitotemporal fibers of association as may exist do not form any compact bundle which can properly be termed the inferior longitudinal fasciculus.

2. There exists a long fronto-occipital tract of association, which medially, represents the fibers spoken of by Déjerine and others, as the inferior longitudinal bundle and which, externally, represents the fasciculus occipitofrontalis inferior of Curran.

3. That the term "inferior longitudinal fasciculus" should be dropped and the term "fasciculus occipitofrontalis inferior" should be used to describe this long association tract.

4. The fasciculus geniculocalcarinus of Archambault is a definite thalamocortical projection tract not to be confused with the fasciculus occipitofrontalis inferior.

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ABNORMAL MENTAL STATES ENCOUNTERED IN A DETENTION PRISON *

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In New York, when a person has been apprehended, or has given himself up, after the commission of a crime, he is lodged in a cell in a police station, for a period of time varying from a half hour to two days. From there he is taken before a police magistrate for a preliminary hearing. If prior to, or during, his arraignment in the magistrate's court, the person shows gross evidence of mental disease or grave physical injury, he is sent to a city hospital (prison ward), where he may be detained for examination, observation and treatment. Otherwise he is committed to the city prison to be held for a further hearing, for the grand jury, for sentence or for trial. Each borough has at least one such prison, known as the detention prison. Here the accused may stay from ten minutes to a year or more (18 months), depending on various circumstances, such as the nature of the offense charged, whether bailable or not, and if bailable, whether bail can be obtained or not. Suspicious persons are detained for investigation as to their reputation, previous criminal record, or to give the police department opportunity to obtain further evidence against them. In cases of felonious assault, abortions, etc., the accused may have to stay for months to await the final outcome of the injuries inflicted on the victims. Persons convicted of less serious offenses are sometimes sentenced to a detention prison for terms varying from a day to a year.

The population of one of these institutions is, therefore, made up of sentenced persons, of those convicted but not sentenced, and of those accused but not convicted. No one under 16 years of age can be committed to a detention prison. In the Brooklyn City Prison, where I have been the medical officer for the last fourteen years, we have annually a transient population of from 15,000 to 20,000, the daily census being about 320 males and 40 females. The ages of the inmates vary from 16 to 85 years, with the greatest number between 20 and 30 years. All races, creeds, colors and nationalities, as well as every occupation and profession, are represented.

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The offenses include every crime against the state, nature, person and property; they include *mala in se* (crimes involving moral turpitude) as well as *mala prohibita* (misdemeanors), violations of corporation ordinances such as the disobedience of the "8 foot" motor vehicle law, or peddling without license, and also violation of federal laws. This, roughly speaking, is the nature of the clinical material with which the prison physician has to deal.

On entering one of these institutions, an observer can at once conclude that there is no such thing as a physical criminal type. According to Lombroso and his followers, an anthropologic examination alone should in most cases be sufficient to detect a criminal, and to determine the particular crime which such an individual is most likely to commit. While it is true that a physical examination of the inmates of one of these institutions will disclose that a great many of them bear stigmas of degeneration, such as sharply pointed skulls, flaring foreheads, low and narrow foreheads, deformed ears, prognathism, notched and widely separated teeth, high arched palates, hare lips, saddle-shaped noses, spinal deformities, clubbed feet, nystagmus, albinism, stammering, polydactylism, syndactylism and hypothyroidism, the number of persons presenting these defects is not greater than one would ordinarily find among the free population.

Dr. C. P. Goring, physician to the Pankhurst Prison in England, undertook a most exhaustive study of 4,000 male convicts. He compared the physical characters of different kinds of criminals with one another, and criminals as a class with the law abiding public engaged in similar occupations. He concluded that there is no physical criminal type. From his measurements, it appears that physically there is a wider divergence between the average Oxford University graduate and the average Cambridge University graduate, than between the criminal and either of them.

Some early criminologists asserted that all criminals are insane. Lombroso succinctly states that "criminality is insanity." Modern penologists oppose this view. T. Mott Osborne, in discussing this subject in a series of lectures delivered at Yale, says:

Most convicts so far from being naturally stupid or showing retarded mental development, are possessed of a keenness of wit that outsiders may well envy. There is, of course, no uniformity of mental attainment or ability; and a one-sided development is often plainly noticeable, some senses like that of hearing becoming abnormally acute. But the talk of any large proportion of convicts being mentally deficient is sheerest nonsense. Another cause of misunderstanding is found in the marks of stupidity, hypocrisy and falsehoods which are adopted by convicts as a means of protection, just as Nature supplied to many animals and birds certain marks and coloring for the purpose of escaping their enemies.

The medical inspector's report in the British Blue Book (1894-1895), states that among the prison population the rate of insanity is not less than three times as great as among the *general* population of the same ages. This higher percentage he believes to be due to the sources from which the average prison population is drawn. He calls attention to the fact that there are many lunatics wandering about the country, who will not be recognized as lunatics until they commit some crime which brings them within prison walls. Glueck says, "In a prison, mental disturbances, not necessarily committable psychotic conditions, exist in a ratio of about 10 to 1, as compared with the incidence of mental disease among the free population." Of 1,000 offenders studied by Spaulding and Healy, 65 per cent. had no discoverable mental defects or aberration.

In studying problems of this kind, statistics are of little or no value. In these institutions, a man charged with homicide for killing another human being by accident is, for statistical purposes, grouped in the same category as a man charged with killing for purposes of robbery. The average person considers as a criminal one who has committed a crime involving moral turpitude. Prison records, in general, however, speak of all inmates of prisons as criminals. Prof. Mayo Smith is correct when he says, "Statistics as to crime and its incidence present the most complicated and difficult problem within the scope of science." I agree with Osborne when he says, "Prison is as full of diverse personalities as the outside world; it is populated by the weak, the gay, the talented and the ignorant. Many prisoners have unusual personalities." Taking all the facts into consideration the conclusion is inevitable that the number of mentally abnormal persons in prisons is unusually large; but whether the percentage is 3, 10 or 35 is purely a matter of speculation.

For the purposes of studying mentality as a factor in crime, the prison population may be divided roughly into four large groups: (1) the accidental criminal; (2) the occasional criminal; (3) the insane criminal, and (4) the habitual criminal.

I realize fully that this grouping is crude and lacks most of the requisites of a scientific classification. None of these groups possesses sharp boundary lines, and as the discussion advances it will be noticed that they overlap. This classification, however, will best serve the present purpose, which is that of an orderly discussion of the subject matter at hand.

I. THE ACCIDENTAL CRIMINAL

The accidental criminal shows, as a rule, no defect of intellect or character, except that emotionally he is somewhat unstable. Therefore, he is called by some the criminal by passion. He is easily excited and

under the influence of alcohol or some temporary mental stress may commit assault or even murder. In reality he is not a criminal. His previous reputation generally is good. He never commits a crime against property, and never for gain or revenge. Numerically, he plays a comparatively minor rôle in forensic psychiatry. The only problem he presents to the prison physician is that after he is committed, it becomes very difficult for him to adjust himself to his surroundings, and he frequently develops a psychosis described in the literature as a prison psychosis or Ganser's syndrome. This is characterized at first by great anxiety and later by clouding of consciousness and amnesia with "islands of memory." Kraepelin interprets the clouding of consciousness as a manifestation of repression. The prisoner is overawed by the impending danger and overcome by the humiliation attending his arrest, and the clouding of consciousness is an attempt to exclude the painful impressions and reminiscences from consciousness.

II. THE OCCASIONAL CRIMINAL

These show no gross defect of intellect, but are possessed of little will power. Their power of discrimination is also below par. On account of their great suggestibility, they yield most easily to temptation. Their instability of character is pronounced. They begin their criminal careers when quite young. They are subject to emotional outbreaks, which are temporary in nature, and it is during these outbreaks that their antisocial activities are greatest. Their reactions are excessive and out of proportion to the nature and extent of the stimulus. They commit crimes against nature, person and property. These criminals constitute approximately three fourths of the "detention" prison population. Long sentences to penal institutions develop the criminal instinct in them and make habitual criminals of many of them.

Under this heading is included also the criminal by "adventure." He begins his criminal career in early youth, having gained inspiration from reading some startling dime novels or seeing a bold "hold up" in a motion picture. Usually he begins by stealing some money or jewelry from home and starts out to seek adventure and to satisfy his curiosity. After he is stranded, he steals from strangers in order to work his way home. On his way, he is arrested for petty larceny or vagrancy and usually lodged in prison, where he comes in contact with hardened criminals. On his discharge from prison, he reestablishes their acquaintance and thus the way is paved to habitual criminality. They are all unstable, changeable and unreliable. They cannot adapt themselves to life. When not in prison, they change their residence and occupation frequently for no apparent reason. Some of them are dull, listless, lazy, shiftless, always following the line of least

resistance. When they do not steal they beg. They are the hobos, vagabonds and tramps, so frequently encountered in county jails and lock-ups. In the higher classes of society they are the dilettantes. Rarely committing crimes requiring violence, they are petty larceny thieves, sneak thieves, pickpockets and forgers. Many of them have dementia praecox personalities, and differ from the psychopathic type (to be noted later) in that they have no defect of intellect.

III. THE INSANE CRIMINAL

This group constitutes a small fraction of the prison population. By an insane criminal I mean a person who, as a result of a psychosis, commits a crime. This group may be separated into two subdivisions: (a) the committable, and (b) the noncommittable.

Among the committable cases are the deteriorated paranoiacs, who commit murder or arson and who justify their misdeeds on the grounds of their delusions. The manic-depressives commit murder, assault or arson during an episode of excitement, or, while depressed, they commit murder or attempt suicide, or both.

Abnormal mental states, due to acute toxic or infectious conditions (except those of alcoholics and drug addicts), are rarely encountered in detention prisons.

The most difficult problems in forensic psychiatry are presented by the periodic insane and the epileptic who commit antisocial acts during an episode of insanity or during a preparoxysmal or postparoxysmal state.

Among the noncommittable cases I include persons with paranoid states without deterioration, the querulous and the militant women. This subgroup is much more common than the one just described. At any time during incarceration any one of these cases may deteriorate and become committable.

Many prisoners with paranoid states have delusions which appear while awaiting trial, especially if they are without relatives or friends interested in them. Having been deprived of liberty and having no one to whom to open their hearts, they become subject to marked depression with delusions and auditory and visual hallucinations. These cases are similar to the induced paranoid states and prison psychosis described by Ganser, Petery and others. Some prisoners develop religious delusions and spend hours and days kneeling and praying. In other cases, the content of the delusions is sexual. The chronic delusional insane are similar, their delusions are more systematized and develop gradually. The accused believes himself to be a great composer, painter, poet or inventor; his neighbors are jealous of him and therefore are his enemies; they are about to rob him of the fruits of his labors;

they are attempting to patent his invention; this leads to a quarrel, which results in an assault, which finally leads to his arrest.

Another is a prophet. He hears the voice of God and sees angels in heaven. Some of these prisoners are hypochondriacs. They feel the brain-softening and the head empty or filled with decomposing insects; the skin has turned to parchment, it is cracking, and the joints are dismembered. Like the neurasthenics they attach the greatest significance to the slightest physical disease or injury. Their ideas are being influenced and drawn out by electricity. The devil is within them, and compels them to say words and commit acts, which they know are wrong. They have no power of criticism.

The greater number of querulants are such on admission. Others, after they have been in prison for some time, laboring under great strain and suspense, begin to doubt the justice and righteousness of the whole world and thus develop into typical querulants. The exciting cause, in most cases, is some unsuccessful litigation. As a rule they are irritable and moody and cannot interpret facts correctly. They fight continuously for their alleged rights, act as their own counsel, examine opposing witnesses and sum up the case, or, if counsel is employed, continuously advise him how to conduct the case. They are very dramatic. Often they draw their own briefs, write lengthy communications to the judges, governor, and even the President. They complain bitterly of the partiality of the presiding justice, and accuse the district attorney and the police department of "framing them up." While fighting their own cases, they hope to improve the laws for general mankind. They sacrifice all their possessions to gain the slightest legal technicality. They are busybodies, continuously advising other prisoners to change lawyers and how to conduct their cases. Very troublesome prisoners they are, always finding fault with the discipline, the food and the medical officers and continuously complaining to the higher prison authorities and making numerous foolish suggestions for the improvement of prison management.

Among the women, a similar type is observed, the so-called militant woman. She insists that nobody appreciates or understands her, and asserts that every one abuses her for no cause whatever. She is fearless, very aggressive and quarrelsome. Any one who disagrees with her is her enemy. She loves power and stops at nothing to obtain it. Apparently there is no defect of intellect. Although these patients lack insight, it is very difficult early in the disease to show the presence of delusions. They are impulsive in thought and action and Southard believes that their tendencies have some relation to sadism. They are usually arrested for disorderly conduct, malicious mischief or assault. Many of the women sent to prison as common scolds are of this type.

They are obstinate, selfish and disobedient. Discipline seems not to have the slightest effect on their conduct.

The acute prison neurosis of the anxiety type, described by Yawger of Philadelphia, we see very frequently among sentenced prisoners, just prior to their parole or discharge, and among prisoners awaiting trial or the result of an appeal. It has been found by Yawger in one third of the convicts coming up for discharge. He believes that a true psychosis does not develop because the mental strain does not last sufficiently long. Our experience bears out that of Yawger, in that the neurosis occurs mostly in persons of constitutional inferiority and in prisoners who have been charged with sexual crimes. The latter, as a rule, are masturbators, and just prior to their discharge they begin to fear that by excessive masturbation in prison, they may have lost their manhood. In most cases, particularly in those who are not habitual criminals, the anxiety is due to worry as to how they will be received by their old associates and how they will be able to make a living. The subjects of this neurosis become very restless, suffer from insomnia, loss of appetite, constipation or diarrhea; their superficial and deep reflexes are exaggerated, and the pulse is very rapid. They smoke excessively, develop tremors and are talkative, irritable and anxious. Many of them have vasomotor disturbances and may lose considerable weight.

IV. THE HABITUAL CRIMINAL

This group may be subdivided into: (A) the instinctive criminal, (B) the professional or incorrigible criminal, and (C) the feeble-minded criminal.

A. The Instinctive Criminal: This class is composed of those commonly called "the prison rounders." They are moral monsters, selfish, brutal, crafty and never show remorse for their misdeeds. Although very obstinate along certain lines, they never display this obstinacy towards those whom they consider their masters. They recognize no moral responsibility, but show no evidence of defect of intellect. They are the thugs, the gunmen and the gangsters who infest the slums of the city. They resort to criminality as a sole means of obtaining a livelihood, commit crimes against the state, person and property, and derive an additional income from the proceeds of prostitution into which they force their feeble-minded female companions.

B. The Professional Criminal: His character and personality are not unlike those of the instinctive criminal, but he is more conceited and more resourceful. He represents the aristocracy of the criminal world. He is intelligent, skilful, ingenious, an accomplished liar and he is very treacherous. He recognizes no obligations to law, society,

parents or friends. He commits murder, burglary, robbery and larceny, engages in counterfeiting and receives stolen goods. Only "big jobs" appeal to him and he plans them with painstaking care. When he meets resistance, he does not hesitate to maim or even murder. His defect is not one of intellect but of character and morals. His bravado is beyond description or belief. In prison he is quite at home, and even there he is busy planning new crimes.

Recently I had an opportunity to study a renowned member of this group:

G. F. H., aged 27 years, an American by birth, a high school graduate, of good family, whose previous and personal history was uncorroborated, was convicted of murder in the first degree and is awaiting execution in Sing Sing prison. He killed two employees of a savings bank in Brooklyn, while engaged with a companion in a daring bank robbery in broad daylight. The robbery netted the pair \$13,000, which they stole while the bank was full of depositors and every employee at his post. G. F. H. was apprehended some months afterward in the far West, where he had been arrested for having killed a pal of his after a quarrel. This bandit had been plying his nefarious vocation for ten years with absolute disregard for human life or property. He admitted killing three men and shooting six others before he committed the last crime and he confessed to having robbed thirteen banks. He appeared refined and intelligent. He never expressed the slightest regret for his cold-blooded murders. Physical, mental and neurologic examinations were negative. He had read quite extensively and was well informed.

Instinctive and professional criminals are sometimes discussed in criminologic literature under the heading "incurable." They have neither desire nor intention to forsake their vicious course. Reformation of habitual criminals of this type is a hopeless task.

C. The Feeble-minded Criminal: He really belongs in the psychopathic group. Southard would say that most of the members of this group are suffering from some form of "psychopathsia." He insists that an attempt should always be made to place these defective delinquents among the hypophrenics, the epileptics, the schizophrenics, the psychopathic monomaniacs, etc., and he warns "not to prejudge the situation in criminology by terming all defective delinquents psychopathic personalities." He wants to "leave room for criminals that are not psychopaths" (professional criminals).

In this group are included all persons who come in contact with the law on account of defect of intellect as well as of character, the latter being the result of the former. This is the most heterogeneous group in our classification. I have no doubt that many will find fault with my inclusion in this group of such cases as are obviously psychotic, cases of hyperthyroidism and other cases of endocrine disturbance and the alcoholics and drug addicts. This criticism is just, but these cases,

are included in this group for want of a better one. The psychotic cases in this group are borderline cases, and the delinquent drug addict and delinquent alcoholic are included because, in our opinion, they are such on account of defective mental make-up and personality.

In this group of defective delinquents, we include the cases of so-called constitutional inferiority—"the misfits of society." They have been defined as "individuals who are possessed of innate mental abnormality, more or less permanent, which has affected their character and mental make-up in such a manner as to have interfered with their proper development, which does not enable them to exist in the community in which they may be placed and to observe sufficiently well the moral and social laws by which we all are governed." Such a constitution need not necessarily be congenital; it may be acquired from accidents of birth, from severe trauma, infectious disease or intoxications. The acquired cases may or may not show physical evidence of disorder of the mind or disease of the nervous system. Scholtz's classification of constitutionally inferior children has been adopted in grouping the defective delinquents found in prison:

(a) *The Indolent Type*.—These are dull and apathetic, lacking interest and enthusiasm, are lazy and shiftless, not unlike the hebephrenic cases of dementia praecox. They tramp through the country and beg, or steal as the opportunity occurs; but usually steal only enough to satisfy hunger.

(b) *The Depressed Type*.—Some one has properly said, "These are never happy except when they are unhappy." They are pessimists and believe themselves "good for nothing." To get along in the world at all, they need some artificial stimulation, so they resort to drugs or alcohol or both. It is then that they become antisocial. They are most pitiable subjects when deprived of their artificial stimulation. Many of the suicides in prison occur in this class.

(c) *The Maniacal Type*.—They display increased psychomotor activity; they are either inattentive or attention wanders rapidly from one thing to another. They have no defect of orientation or consciousness, no delusions and they are always very busy, but, as White says, "There is no orderly consecutiveness in their acts." They are usually in prison because of disorderly conduct, malicious mischief or minor degrees of assault.

Cases of pure depression or pure mania are rarely seen in detention prisons. The mixed forms, cases of so-called circular insanity and cyclothymia and hypomania are quite common. During the stage of depression, they present no evidence of mental retardation, delusions or hallucinations and have a definite, but not always correct, insight. The depressed state may be preceded or followed by a mild form of

excitement during which the subjects appear to feel unusually well; they are very bright and unusually loquacious. When ordered to do some work, they do it better and more quickly than any one else. This type of person, when free, engages in all sorts of projects quite foreign to his natural activities. Hecker (cited by Jelliffe) relates an instance of multiple marriage engagements made in the euphoric stage and broken in the depressed stage. Many persons charged with polygamy and seen in detention prisons are cyclothymics.

Gregory describes cases with transient attacks of manic-depressive insanity, which are characterized by short attacks ranging from a few hours to several days but which are difficult to recognize unless they are accentuated by some exogenous factor, such as alcohol. These fleeting attacks when mild in type and unaccompanied by alcoholism are often mistaken for hysteria, psychic epilepsy or migraine, and when they are accompanied by alcoholism are often wrongly interpreted as mental states due to alcohol. During these attacks the patients may commit sexual crimes, assaults, suicide or homicide.

Circular or periodic insanity is of medicolegal importance because many criminal lawyers, when they have no better defense, plead that the accused committed the crime charged during a temporary psychosis and therefore cannot be held responsible.

(d) *The Impulsives*.—These are prototypes of the criminal by adventure. As children they have the impulse to run away from home; in later life they become vagabonds, tramps, prostitutes, kleptomaniacs, pyromaniacs, dipsomaniacs, etc. All of them exhibit a perversion of feeling which has a peculiar effect on their habits, temper and conduct in general. The impulses appear without cause and their accomplishment is followed by a feeling of relief. As children, they exhibit great cruelty toward animals, and as adults, when they commit crimes, they do so with fiendish brutality. They are found in prison charged with every crime on the statute books, particularly with those against sex. They may be capable of considerable training and are therefore always a menace to society.

(e) *The Imperatives*.—In the imperative, the feeling of compulsion is coupled with doubt or fear, or both. This type, therefore, is not common among criminals.

(f) *The Pathologic Liar*.—Healy says, "Pathologic lying is a falsification entirely disproportionate to any discernible end in view, engaged in by a person who at the time of observation cannot definitely be declared insane, feeble-minded or epileptic." Pathologic liars give a history of bad heredity, poor environment and poor memories. They are emotionally unstable and have no idea of exactness or precision. Their attention can easily be attracted, but cannot be held for a rea-

sonable length of time. They are very superficial, take no precautions to prevent their lies from being detected and most of them have no motive for lying, perhaps believing in the truth of their own lies. They are high grade mental defectives and never wilfully resort to criminality. Owing to their auto-erotism, their crimes are frequently sexual.

(g) *The Epileptic*.—The epileptic who has not sufficiently deteriorated to be considered insane is frequently the subject of medicolegal inquiry. Many epileptics are arrested for intoxication, when, after recovering from a convulsion, they cannot give a coherent account of themselves. In some of these cases, it may be impossible to determine whether the convulsion is due to epilepsy or to alcohol, especially when the previous history is unobtainable. Epileptics are often brought into prison charged with disorderly conduct or endangering the morals of minors, when during a state of temporary confusion, after a convulsion, they perform automatic acts, such as undressing in public or exposing their genitals. In prison one often meets persons charged with crimes of violence and brutality, committed during an epileptic equivalent. This state is followed by total or partial amnesia, and may or may not be associated with or followed by typical seizures.

(h) *The Perverts*.—Sexual perverts are found in large numbers in all prisons. The masturbator with his typical neurasthenia and hypochondriasis is the most common. Exhibitionists and *voyeurs*—the former charged with endangering the morals, the latter with having in his possession obscene pictures—are quite common.

(i) *The Prostitute*.—Although not invariably feeble-minded, she is almost never a normal individual. She is devoid of self respect and possessed of mental and moral inertia. Indulgence in alcohol and narcotics follows rather than precedes prostitution. (The same may be said of criminality.) About 60 per cent. of those we see have a mental age of 10 or under.

There is a type of prostitute known as the "degenerate woman of the higher classes," or as Lombroso calls them "prostitutes of society." The historic cases of Mmes. Steinheil, Tarnowska and von Schoenheck are examples of this type. These women are highly emotional, inconsistent in thought and action and markedly inclined to lying and intriguing. They are abnormally suggestible, have very poor judgment and are utterly devoid of morals and ethics. Their innocent childlike playfulness, attractiveness, fine manners and superficial culture have a fascinating influence on men high in social, financial and professional circles, who are easily ensnared by them. They are in their ways very shrewd and cunning and thus manage to keep out of prison. They

resort to various forms of blackmail, to which their victims readily submit rather than to go to law and gain unpleasant notoriety.

(j) *Kleptomaniacs*.—Kleptomania is classed by White among the psychasthenias, but he adds that both kleptomania and pyromania may form part of a psychosis. Kleptomania is not to be confused with the automatic stealing of the epileptic or the habitual stealing of the feeble-minded. Kleptomaniacs have impulses to steal; they do not need what they steal and can well afford to buy what they steal. Most of them are women, and often they steal objects which cannot be interpreted, even by one with a wild imagination, as sex symbols. They usually commit these crimes near, during or immediately after a menstrual period. The behavior of girls and women charged with crimes which are committed before or during the menstrual period is so peculiar and so different from others, that we are led to believe that this fact must have some etiologic significance and is more than a mere incident.

Abnormal mental states due to dysthyroidism are most commonly seen in women suffering from hyperthyroidism. They usually are committed, charged with quarreling, disorderly conduct, assault, malicious mischief and homicide. They present the clinical features of ordinary hyperthyroidism. Owing to their irritability and quarrelsomeness, they are very difficult prisoners to manage.

(k) *The Alcoholics*.—Since prohibition has gone into effect, alcohol is beginning to be a negligible factor in crime. Prior to this period, the occasional drinker came in conflict with the law while acutely intoxicated. He was usually arrested because, during the second stage of drunkenness, i. e., the stage of excitation, he committed some breach of the peace.

The chronic alcoholics were of far greater medicolegal importance. Many of them were brought in with typical delirium tremens. Some of them developed the delirium from one to eight days after admission. When arrested during the delirium, the most common charges against them were, malicious mischief, assault, attempted suicide, murder or arson. Some of our chronic alcoholics seemed to have survived several attacks of delirium tremens without showing any signs of mental deterioration. Most of them, however, and this applies also to those who never had delirium tremens, were found to have progressive weakness in the intellectual and ethical spheres. Their sense of morals was on the decline. Memory and orientation were poor, and inhibition was almost nil. The power of apperception was dull and they were subject to delusions, the most characteristic being of jealousy and marital infidelity. It may be said that those having delusions of jealousy or marital infidelity are most likely to commit murder or suicide, or both, at the same time. Those whose moral sense is on the decline

commit forgery, embezzlement, larceny, or write threatening letters for purposes of extortion and commit sexual crimes.

Dipsomaniacs, during their drink periods, commit forgery, steal and write threatening letters, which, after they have ceased drinking, they do not remember at all. The subjects of this disease all gave a history of neuropathic antecedents. Kraepelin, Gaupp, Aschaffenberg and others consider dipsomania a larval form of epilepsy. It is characterized by a premonitory period of depression, retrograde amnesia and amnesia for the attack itself. True dipsomania must be differentiated from so-called periodic drinking, which may be a phase of some psychosis or an expression of some form of disordered personality, such as the mildly paranoid or jealous type or the hysterical personality. The prognosis and treatment of these cases depend on the underlying cause.

(1) *Drug Addicts*.—Most delinquent drug addicts are burdened with a degenerate heredity. About 90 per cent. of them are feeble-minded. If not arrested for violating the law governing the use and possession of narcotic drugs, they come in conflict with the authorities during the period of forced withdrawal of the drug. It is then that they commit petty larceny or burglary, usually in the second degree, i. e., in the daytime and without force. Sexual crimes, owing to their early impotence, are not very common among them. In our earlier experience, it was rare to find a drug addict charged with a crime of violence, except in recidivists who had already been in a state prison, penitentiary or reformatory, and while there, had acquired the drug habit. Recently, however, it seems that drug addicts commit crimes involving violence, but they require unusually large doses of their necessary drug prior to the actual commission of the crime. Crimes of violence among the colored addicts are comparatively rare. This is probably due to their innate cowardice.

To use morphin, opium or cocain seems now to be out of fashion. Heroin "sniffing" is the latest style. Morphinists are characterized by their pathologic secretiveness, cowardice due to lack of selfconfidence, lying and seclusion. Any of these drugs, when taken in considerable quantities over a long period, produces a distinct change of personality. The addict becomes morally dull, and although memory and orientation may be preserved, he has a tendency to confabulate. We have seen more cases of Korsakoff's syndrome in heroin and morphin fiends than in alcoholics. Many an unjust criticism of institutional management owes its origin to the lies circulated by this class of prisoner.

The number of cocain users today as compared with five or six years ago, is almost a negligible quantity. Excessive users of cocain suffer from acute hallucinatory confusion and mania, and the withdrawal period is likewise often marked by most violent mania, with

delirium, which may persist after craving for the drug has ceased. In rare instances instead of excitement, there may be depression with hallucinations, usually visual in nature.

The Chinese never use cocain or heroin and rarely morphin. They resort chiefly to "opium smoking." They do not develop psychoses as frequently, nor do they seem to suffer as much during the period of withdrawal, as the white and colored races. Dr. Ellis, who observed the same clinical variations in the prison at Singapore, believes that this is because the Chinese addicts are very destitute, and the drugs which they can afford to buy are impure and adulterated. This reason cannot apply to the Chinese observed here, because they use the same quality of narcotic that the whites do. We are inclined to believe that the difference in symptomatology is due to racial differences.

Most drug users eventually become markedly deteriorated. The degree and type of deterioration depend on the mental make-up and personality of the user, the kind of drug used, its amount and the duration of the habit.

(m) *The Hysterical Type*.—These persons exhibit an unstable mental equilibrium, and are easily influenced by suggestion. They are subject to phobias and obsessions, states of automatism and dream states with confusion and clouding of consciousness. It is during these abnormal states that their antisocial activities most commonly come to light. An hysterical person is likely to commit any crime, especially those involving violence, when subject to any of these states. A purely hysterical person, unless he is in addition burdened with the criminal instinct, rarely commits a crime against property. The diagnosis of hysteria is as uncertain in the prison population as in the free population. In the absence of hysterical convulsions, sensory changes, constricted visual fields, paralyses and contractures, amnesia and episodic phenomena, a positive diagnosis may be impossible. Amnesia with "gaps" of memory, which are not the same on going over the same ground, may be of some aid in the diagnosis of hysterical amnesia. The course of all mental disorders associated with hysteria is extremely variable. The unusually large number of defective personalities and characters found among criminals must always be borne in mind when one attempts to ascribe the cause of an antisocial act to hysteria.

The freudians assert that they have achieved most brilliant results in the treatment of criminals with hysterical personalities. Indeed, they assert that they have solved some of the most difficult problems in criminality quite easily and satisfactorily, at least from their point of view. By psychanalysis, they have found that owing to numerous unconquerable obstacles to the gratification of overwhelming sexual impulses the person is put to the difficult task of sublimating his wishes

and instincts or suppressing and repressing them. In the unconscious, then, the repressed incest complex, inversion and perversion of complexes, become charged with newly repressed energies, in consequence of which the person is compelled to satisfy his perverse wishes, and to do something that society and morality have forbidden, i. e., he commits a crime. Psychanalysts admit that environment, education, physiologic and economic factors share some responsibility in the evolution of the criminal, but these, they assert, are of only minor significance. From their point of view criminality is the expression of a neurosis, of an impulse to do wrong. Criminality to them is repressed sexuality or an equivalent thereof. This, in brief, is their explanation of kleptomania, pyromania, dipsomania and allied disorders. Any one who comes in daily contact with criminals, and knows what liars and phantasists they are and how easily they lend themselves to all sorts of suggestion, is inclined to be very cautious and rather skeptical in the evaluation of symptoms and in the drawing of conclusions from uncorroborated statements which one obtains from them.

The subject of simulation in criminals is a very important one to the prison physician. According to White and others, the attempt at simulation is in itself a type of reaction which is an indication of defective personality. In many instances, the clinical picture of the "alleged" or "genuine" psychosis will not be decisive, and the social as well as the legal aspect of the case has to be taken into consideration. The more intelligent the prisoner the more difficult is the problem. As a general proposition, it may be said that one who is charged with murder, rape or arson is more likely to "play insane" than one who is charged with larceny, the lesser degrees of assault or injury to property. Although intentional deception may easily be recognized, it is more difficult to exclude a genuine coexisting mental disorder. Some of the most common mistakes simulators make are: (1) exaggeration of individual symptoms, (2) absurd and entirely wrong answers to ordinary questions, (3) wrong combinations of symptoms of different psychoses such as epilepsy and manic-depressive insanity, and (4) a too sudden onset or recession of symptoms.

In mild genuine mania, as a rule, there is some connection of thought with the surroundings; in simulated mania usually there is none. Too much incoherence in a delusion should always arouse suspicion. Simulators invariably attempt to impress on the examiner that they feel "queer" in the head, that they "hear voices" and "see things." This is particularly the case if the prisoner has had some prison or asylum experience. The onset of psychic disturbance immediately after arrest, during trial or after conviction but before sentence is strongly suspicious of simulation.

The prisoner's conduct during the examination is of great aid in the diagnosis. The malingerer avoids looking the examiner in the face, and it is quite evident that he is able to follow the conversation because he soon manifests a symptom, which the examiner tells those near him is missing. The symptoms are exaggerated while the examination is in progress. The prisoners threaten to commit suicide, but never attempt it. The physical concomitants of mental disease such as changes in the circulation, digestion, vasomotor disturbances, loss of weight, changes in the reflexes, pupillary changes and insomnia, are lacking. The subjoined case is typical of the numerous cases of simulation which have come under my notice.

M. D., an Italian shoemaker, aged 31 years, totally illiterate, with good family and personal history, was committed to the Brooklyn City Prison, charged with murder. He shot and killed his mother and his sister, the latter with her baby in her arms, for the purpose of appropriating \$35 which he knew his mother had saved and hidden in a cupboard. After he had been in prison several weeks, and the seriousness of the case began to dawn on him, he began to "play crazy." At first, he refused to eat and was depressed, but after twenty-four hours apparently he became hungry, ate and became maniacal. During one of the numerous examinations, he did not recognize a button, a button-hook, a knife, a match nor a watch, when these articles were shown to him. Three and 4 he said were 8; 2 minus 2 he said was 3; he could not repeat two figures; he did not know how many fingers he had, what a spoon was used for nor his name. To all questions as to the present or past, he answered, "I don't remember, I don't know." The examination was interrupted by the prisoner several times because he said he had headaches, was "all mixed up" and "blood was rushing to his head." When tested for the Romberg sign, it was found to be absent. After the examiner had assured the attendant that something was wrong because the prisoner did not sway his body, he found on retesting him that he could not stand at all, but fell in all directions, with his eyes open as well as closed, and with the feet separated. On one occasion while the prisoners were exercising in the corridor, he had a convulsion during which he was extremely careful how he fell; and the administration of 30 drops of rhubarb and soda by mouth, which he opened voluntarily after the first request, was followed by disappearance of the convulsion. In the prisoner's presence, we instructed his keeper that should he have another convulsion, he was to be put into an ice cold tub bath and later into a padded cell. He never had another convulsion, although he stayed with us about three months afterward.

There was never any doubt in our mind that this man was simulating insanity, in order to cheat the death chair. The nature of his crime, the puerile way in which he simulated insanity, and his general mental make-up, with the absence of signs of organic, mental or nervous disease led us to believe that he was a moral imbecile, and from the fact that he knew enough to sham insanity we thought we were justified in our conclusion that as the law now is, he had sufficient knowledge to know what he did, and that what he did was wrong. He was in due time tried, convicted and sentenced to die in the electric chair. The conviction and sentence were both upheld by the highest court of the state.

TROPICAL NEURASTHENIA, TROPICAL HYSTERIA AND SOME SPECIAL TROPICAL HYSTERIA- LIKE NEUROPSYCHOSES

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These clinical notes, made during my residence in the tropics, are submitted simply to supplement the standard literature available to Occidental students. The disorders noted are among the most prevalent and baffling, as well as the least understood, of all problems to be faced by the physician and pathologist in tropical work.

NEURASTHENIA

Although the great prevalence of neurasthenia is a matter of common knowledge to physicians practicing in the tropics, the literature on the subject is not extensive. King, writing from Porto Rico, states that comparatively few persons entirely escape this disease if they live in the tropics for any length of time. Methods for its prevention had to be instituted for the benefit of foreign employees during the building of the Panama Canal. Fales, who was one of the first to give tropical neurasthenia special consideration, states that it is not only prevalent in the Philippines, but is also an important factor in preventing acclimatization of white men in the tropics "and will prevent successful colonization of the Philippines by Americans." Castellani considers the disease to be extremely common all over the tropics, in Europeans as well as in natives of the educated class. Plehn recognizes its wide distribution and gives its ravages as the principal reason why Occidentals should not attempt to reside permanently in the tropics.

In fact, all writers who have given the subject serious consideration recognize tropical neurasthenia as one of the important diseases of warm countries. As a primary disease, but more particularly as a secondary state, it is extensively prevalent among people of all ages and both sexes, foreigners and educated natives, in all parts of the tropical world. As a problem in tropical pathology it has distinctive features, and it is with these features that we are particularly concerned.

Fundamentally, neurasthenia is the result of a person's inability to stand the stress of life to which he is subjected, shown by excessive fatigue and irritability. This may be due to congenital and developmental deficiencies or the symptoms may be superimposed in other diseases. Both types have a high incidence in the tropics.

Etiology.—In addition to the accepted etiologic factors, there are those peculiar to warm climates. A study of both native and foreign residents in almost any tropical colony suggests a fertile hereditary soil for neurasthenia. The circumstances which induce many foreigners to live in the tropics often are of themselves indications of some nervous unbalance, shown by the fact that the foreign population of any Oriental city contains more than the average percentage of persons of erratic or unstable nervous dispositions. Few natives are mentally constituted to withstand the normal stress of Western civilization which so many of them adopt in zones under influence of Occidental ideas.

Children and adults are affected. The most striking examples are seen in foreign school children, 8 years of age or older. In these young people physiologic development is taking place in accordance with tropical environment, and we try to train them in accordance with Western ideas. The result is languor, lassitude, headache, irritability and nervous exhaustion to a remarkable degree.

Sex seems important. All observers agree that women are greater sufferers than men, and the highest incidence is found among girls at about puberty. One explanation of the greater prevalence among women is found in the abnormal life; the increased sexual activity; more frequent childbirth or injurious activities to avoid it; the menorrhagias and dysmenorrhoeas so prevalent and weakening, and the greater emotional stress under which foreign women live in most of these far countries.

Race is a striking feature among the predisposing causes. In natives it is usually more prevalent among those who are trying to adopt Western methods of energy, application and efficiency and who are unprepared by breeding, training or environment to do so. This is strikingly illustrated in the Philippine Islands where twenty years of constant effort to establish Occidental methods have led to a decided increase of the disorder among the younger generation of more progressive Filipinos.

Among foreigners in the tropics, the victims of neurasthenia constitute an ever-increasing army of misfits created in the process of acclimatization or adjustment to the immutable demands of an environment for which the white race is unsuited by hereditary or acquired characteristics.

Secondary or symptomatic neurasthenia also has a higher incidence in the tropics than in temperate climates because the variety of underlying diseases is larger and in many instances more obscure. There may be mentioned the vagaries of latent parasitic infections of the alimentary canal and liver; malaria, vasomotor complexes, syphilis, fevers, obscure infections, metabolism and internal secretion disturbances,

organic nerve disease and many others. Anemia of the central nervous system, such a frequent condition in the tropics, is an important cause of symptomatic neurasthenia. Alcohol, tobacco, drug and other habits probably are more potent factors than they are in temperate climates.

The climate, actinic rays of the sun and other physical forces receive their share of blame in most medical discussions of the subject.

Symptomatology.—While the clinical picture in general is similar to that given for neurasthenia in temperate climates, certain symptoms, particularly those expressed as disturbed special senses—stand out with more prominence or are peculiar to the tropical disorder. Disturbances of sight and hearing differ from those of the disease in colder climates, chiefly in that the preliminary stage of oversensitiveness is more defined and harassing. The later stage of exhaustion is more profound, and recuperation is slower. Changes in both taste and smell are as pronounced and annoying as those of the other two senses.

Disordered menstruation, metrorrhagia and dysmenorrhea are exceedingly frequent, annoying and often serious symptoms. These conditions often precede symptoms of neurasthenia though they are not always forerunners of that disorder, but the association is close and significant.

Diagnosis.—As the diagnosis of neurasthenia usually is made only by exclusion, that part of the problem peculiar to the tropics consists in the elimination of the special conditions mentioned under etiology. Frequently this is exceedingly difficult. More unsuspected lesions are first found in the necropsy room than is the case under similar circumstances in temperate climates.

Prognosis.—The disorder is a more serious one for the foreigner in the tropics than it is for him in his home country. Several authors attribute the high rate of insanity among Occidentals more to neurasthenia than to any other cause. Less striking, but equally definite, mental deficiencies follow or develop from neurasthenia with considerable frequency. It is a factor in the difficulties encountered by foreign women in nursing their babies, contributes to the birth of moral and physical weaklings and no doubt, as claimed by several writers, to the eventual degeneracy of a white race propagated in the tropics.

Certain foreigners who are able to adapt themselves and become acclimated recover from the disease without leaving the country, but in most instances a change of climate is necessary to avoid permanent invalidism.

Prognosis for the native is similar to that for an Occidental in his own environment. There is this difference, that "back to nature" for the educated native means such a tremendous sacrifice that it will be made less frequently. The problem of neurasthenic misfits, the place

they will assume and the influence they will exert, socially, morally and economically, is inextricably bound up with the future development of tropical countries.

Preventive Measures.—For the Occidental prevention is found in less work, less food and more sleep than he is used to. The rules for the prevention of the various infections so potent in predisposing to neurasthenia are for the most part well known and not difficult to carry out. It is well for the foreigner to recognize that the natural laws have Oriental variations; that they are unalterable and must be obeyed. For the native, prevention consists in holding his ambitions and energies within his natural bounds and resting before the breaking point is reached.

Certain foreigners should avoid the tropics as a permanent residence. This list includes women, children and old persons, neurotics and those with tuberculous tendencies. Foreigners should have a change of climate with the first appearance of well marked symptoms of neurasthenia. Other treatment is unsatisfactory.

HYSTERIA

In various papers dealing with the tropics and tropical peoples in general there is some mention of hysteria, and the great prevalence of the hysterical character or temperament among such people is frequently noted, but medical literature on the subject is meager. Some writers on tropical neurasthenia include therein much that more properly is hysteria, and some medical writers on hysteria mention that the disorder finds ideal conditions and its greatest prevalence among Orientals. My studies on the subject have been made for the most part among the various nationals living in the Philippine Islands and to a less extent in other tropical and Oriental countries. Like many other cosmopolitan diseases, hysteria in the tropics has certain distinctions and problems which are sufficiently peculiar to warm countries to warrant their brief consideration.

Foreigners.—The reasons for a higher incidence among foreigners than is encountered in their home countries are largely those already mentioned for neurasthenia. Promotion is frequently rapid in colonial services and foreigners may fall heir to responsibilities, business and social, far beyond what they would have earned in their home lands and frequently beyond what they are prepared to assume with ease and dignity. Foreign women advance with their men to positions of social responsibility for which they may not be suited and which require constant effort to sustain. Add to this the environment worries of every kind, from insects to bad water, canned food, housekeeping and the climate and a hysterical temperament which might lie dormant under

ordinary stress becomes active. The clinical types and manifestations of hysteria among foreigners do not, as a rule, differ materially from those seen in other countries.

Natives.—Among the dark-skinned natives, the hysterical character or temperament is all but universal, and the disease itself is amazingly prevalent among all classes. The hysterical temperament, with its childish type of mind as the foundation on which the disease is built, is manifested in a variety of ways. The principal features of negativism and impulsiveness, emotional instability, tendency to be influenced readily, remarkable egotism, love of and tendency to create sensations, the desire to confabulate, to fabricate and to simulate, fertility in planning and promising and delay in execution—all exist as natural attributes. With neurologic elements attuned by stress to harmonize with the psychology and an environment calculated to develop the picture, we have ideal conditions for the propagation of the hysteria which already potentially exists.

Emotional instability in particular is so widespread among the Malays and several other tropical races as to be all but universal. It is seen in children and grown people of both sexes, although more strikingly among females. The ordinary types are characterized by ever changing moods. From smiles to frowns, tears or a *bolo* is frequent and without serious cause. Sensitiveness is extreme and the slightest reproof leads to tears, scorn or violent bursts of anger, and nearly always is followed by an unrelenting grudge. A word or a suspected slight may lead from gaiety to depression. Changes from happiness to misery, from the gay-colored dress to *crêpe*, or from life to self-inflicted death, are frequent. Emotional reactions replace reason, discipline and justice in the control of personal affairs and sometimes those of state. They are the governing force in the rearing of children and influence the making and enforcing of laws. The wife frequently controls her husband and children by them, and men sometimes use them as an asset in business or public life.

Suggestibility reaches a stage of high development, both quantitatively and qualitatively. It is this element in the hysterical character that makes peonage a normal custom, insures one more power in tribe or community rule, and must be materially modified before personal equality or representative government by suffrage will be appreciated or practiced.

The element of sensationalism in the hysterical character is one of the most interesting in tropical pathology, because it explains the love of gossip which has reached such degree of development as to be a constant wonder to a Western student. The "underground telegraph" disseminates information more rapidly than the public press. In order

to produce proper sensation and to allow each retailer to attract attention to his own importance in the matter, exaggeration follows exaggeration with great rapidity. By this method the most trifling incident becomes a great affair. By it character is destroyed and homes ruined to an extent and in a manner all but unbelievable to Occidentals. All of this is done, not with the prime idea of injury, but because of the necessity of personal exaltation.

The motor and sensory manifestations of hysteria and the clinical types, diagnosis and treatment in ordinary cases do not differ sufficiently from those of the disease in other countries to require special discussion here.

SOME SPECIAL TROPICAL HYSTERIA-LIKE NEUROPSYCHOSES

Under this heading it seems appropriate to note some of the neuro-psychoses which have their origin in the hysterical character, find favorable stimulation in a tropical environment and are perpetuated by custom. Strictly speaking, these symptom groups are not so much neuro-psychoses as they are aberrant expressions of disordered neuro-mechanism among persons of a low order of mentality. They are special forms of deviation or expressions of immaturity brought out by influences similar to those which produce hysteria among more developed persons.

They may be arbitrarily classified thus:

Mimic Groups: Latah, mali-mali, etc.

Fury Group: Amak, juramentada, dalahara, misala, head hunting, etc.

Exaltation Group: Flagellate worship, schamanism, certain religions, dancing ceremonies, etc.

Stoical and Depression Group: Hari-kari, etc.

Illusion and Delusion Group: Witchery, poisoning mania, various mirages, etc.

Mimetic and Impulsive Group.—This group of psychoses includes in whole or in part latah, mali-mali, bah-tschi, yaun and myriachit, Gilles de la Tourette's disease. The "jumpers," the "barkers" and the "jerks" appear to have some characteristics of this group on the one hand and of the tics on the other. Even the tics and habit spasms are in close affinity with the group, and the possibilities of combined tic and saltatory spasm in the same patient may explain some of the difficulties in harmonizing the symptomatology.

Etiologically, all these psychoses spring from the same soil of the hysterical character among people of various degrees of mental imperfection and who express themselves in various ways, from the motor reaction in spasm in the true tics to echolalia or other types of pure mimicry, as in latah and mali-mali. In some conditions such as Gilles

de la Tourette's malady, we have both the muscular spasm and the mimicry. While all the types in the mimetic group, as well as various combinations, have their homes in the tropics, the most interesting psychosis peculiar to warm climates is represented in the condition most widely known as *latah* or *mali-mali*. This is a true mimic psychosis characterized by coprolalia, echokinesis and the prompt execution of given orders. All, or any combination, of these characteristics may exist in a given patient. The geographic and ethnologic distribution of *latah* is large. It includes many races, the Malays being particularly susceptible. It is found, however, chiefly among Orientals, mixed breeds and rarely in Europeans residing in the tropics. It is particularly prevalent in the Malay Peninsula, Java, Siam, China and the Philippines.

Women are more frequently affected than men, and the malady usually appears in young persons and continues throughout life. Attacks are ushered in by a variety of stimuli. Usually it is necessary to focus the patient's attention suddenly by a sharp noise, quick movement, sudden touch or flash of light. When the patient's attention is secured, the particular form of mimicry may be continued at the will of the operator without any special concentration. To break the spell, it is necessary only to stop the action being imitated.

The coprolalia frequently is introduced by more or less involuntary disconnected sounds, words, exclamations or obscure expressions. It may be continued as a regular word mimicry or echolalia. The echokinesis consists of more or less perfect motion mimicry, and even the most remarkable gymnastics are carried out to the point of complete exhaustion if the stimulation is continued. The lives of *mali-mali* patients frequently are made miserable by persons taking advantage of their known weakness for amusement. On the other hand, one hears pathetic stories of the death or injury of infants in the charge of "amahs" who suffer from *latah*. Also, authentic instances are on record in which patients with *latah* have been started to fighting and have killed each other. Demonstration attacks of *latah* resemble a mesmerism seance, and the face of the patient has the blank uncertain appearance of one under hypnotic influence. Patients usually have no distinct recollection of what takes place while they are in the *latah* state. One sees patients with incomplete cases of *latah* who, when caught unawares, make the initial movements in sound or action, and then gain sufficient control to throw off the influence.

The disease apparently is incurable, but patients sometimes learn to limit the attacks by careful and studied personal control. *Latah* is not limited to man, and some very good examples of the *latah* echokinesis may be observed in domesticated monkeys. "Having the last word" of more advanced civilization has been suggested as a

deformed evolutionary remnant of the echolalia type of these mimetic psychoses.

Fury Group.—In this group are included entirely or in part amok, juramentado, dalahara, tropenkohler and misala. Clinically, there are two divisions in this group which differ in degree only. Amok, of the Malays, and juramentado, of the Moros, are very similar, if not identical, and the same may be said of dalahara of the Malays and tropenkohler of Africans.

Amok and Juramentado.—Amok is a Malay word which means a frenzied desire to murder. Juramentado is a term used by the Spaniards designating a Mohammedan Moro who, after certain religious rites, undertakes to kill whom he can until he himself is killed. The psychosis is limited to men largely of the Mohammedan faith and ethnologically largely to Malays and Moros. It is reported as occasionally seen in India and Siberia. Among the older theories of the etiology of these conditions, there may be mentioned only to be discarded, opium using, hasheesh, Mohammedan hatred of Christians, alcoholic insanity and epilepsy.

Of more important observations which are not in conflict with the fundamental idea that the condition is a psychosis in a hysterical character, there may be mentioned the conclusion of Wallace, that it is a form of suicide. It is a well-known fact that natives of the Celebes and many other Oriental countries consider suicide an honorable proceeding and a proper way to release oneself from a difficult position. They take the amok method just as a Japanese prefers hari-kari and an Occidental shoots himself. Scheube considers that an important part is played by the want of control of the passions and desires, due to the defective training and education of the Malays, who as a race are abnormally excitable, and the fact that they attach no importance to the lives of their fellow beings.

The exciting causes are various. There may be mentioned grief over imagined or real injustice, infidelity of a wife, death of a loved one, fear of disgrace and particularly accusation of cowardice. The attacks are ushered in in two ways. In one it is preceded by days of melancholic torpor in which the patient becomes morose, gives up work and avoids his fellows. In other instances, and particularly in the juramentado of the Moros, the attacks are brought on by religious rites, incantations, music, dancing and other methods of psychic stimulation. There may be a premonitory stage during which patients state that everything before their eyes becomes red or black, giddiness sets in, and the patient remembers nothing else until the attack is over. In either case, when a sufficient frenzy is reached, the afflicted person suddenly runs into a crowd of persons or through the streets and with his "kris"

or "barong" kills whomever may come in his way, regardless of sex, age, race or any other of the usual considerations of affection or fear. One of these patients will charge into a company of armed soldiers with the same recklessness as into a group of defenseless women and children. If not killed or restrained, the patient may continue his activities from a few hours to days, until they are terminated by exhaustion or suicide. After the attack, in case of failure to secure the coveted death, the patient sinks into a deep stuporous sleep from which he finally rouses in a disagreeable, taciturn frame of mind, which may last for weeks, during which time it is well to avoid the fanatic.

Dalahara, Tropenkohler and Misala.—These conditions in etiology and clinical manifestations are very similar to the fury psychoses, except that they do not go so far. The attacks reach their apex in a spirited word quarrel, and the patients do not come to blows.

Dalahara is the quarreling disease of Filipinos described by Musgrave and Sison; tropenkohler is a similar condition described by Plehn as existing among certain African tribes; while misala appears to be the same condition among Nyasalanders, described by Howard. It is probably prevalent among many Orientals. The affliction is found in both sexes. It is more prevalent among females in the Philippines and among young adult males in Nyasaland. It affects young and old, but in Manila, where it has a high incidence, it reaches its greatest perfection among the ignorant "tao" class of betel-chewing women.

A person afflicted with the complaint will start a quarrel with a relative, friend or entire stranger. She will rapidly work herself up to a perfect frenzy of speech and gesticulation, without apparent object or cause. The attack may last from a few minutes to hours or until exhaustion takes place, only to be repeated again as opportunity offers. This affliction is not the usual manifestation of anger for cause, but a psychosis found only among the most ignorant persons.

The head hunting of the Igorots and similar practices among other heathen people represent methods of warfare and are only practiced on assumed enemies. However, it is well to remember when traveling among such people that all persons not of their own tribe are likely to be classed as enemies.

Exaltation Group.—In this group are included schamanism, ram-aneniana, the dancing mania of Ramisiary, Saint Johns and Saint Guys; the great variety of other religious, funeral, marriage and similar rites of celebration so prevalent throughout the tropical world and wherever ignorance and the hysterical temperament are found.

The object in all of these is to secure a frenzy of emotional excitement, which is attained by incantations, singing, shrieking and dancing, aided by erratic habits. In some of the varieties, a single person may be

the patient, but in most instances the functions are community affairs. Some of the "wonder fakers" take advantage of this inherent quality to propagate frauds, religious and social, particularly in the healing art. An interesting phase in producing emotional excitement is that element which produces self-injury to attain it. One of the best examples of this is seen in the flagellate worship among certain Filipinos. The physical injury (even death has resulted) from the practice of this psychosis was so great that it has been prohibited by the church as well as by law. Persons belonging in the exaltation group are deficient of a harmless variety and our principal concern should be methods of preventing a propagation of their kind.

Depressive Group.—This group may be considered the antithesis of the fury group. It includes the stoics, depressives and certain others who end by one of the forms of self-destruction. An observation of never ceasing interest to physicians practicing in the far east is the apparent ease with which many natives refuse to acknowledge pain and the nonchalance with which they will sacrifice their own lives. Contrary to general belief, stoicism is not a natural characteristic and does not properly belong in the dominant emotional nature of most Orientals. Its extensive practice must be looked on as an attainment brought about by effort just as in the case of some of the other psychoses. It does not have the high incidence among Filipinos and Malays that it does among Japanese, Chinese, Indians and certain other races.

Suicide by a great variety of methods is practiced extensively among many far eastern and tropical peoples. In some places it is an honorable procedure even to the extent of having religious and state endorsement. The Japanese meet the "Honorable Mr. Suicide" by hari kari; the Indian by poisoning; and the Egyptian by strangulation. Among few, if any, is the act thought a disgrace, and with many it is considered an honorable and desirable way out of difficulties.

Delusion and Illusion Group.—Delusion and illusion, with their attendant deceit, mysteries and intrigue, are basic factors in the characteristic Oriental atmosphere. It is distinctive of the East, and has been much written about in prose and poetry. It is the indescribable something that one feels shortly after arrival in a tropical country and which grows to influence, absorb or destroy. It takes many forms, all suited to the normal stage setting in environment and to the emotional natures of the people. Witchery, mesmerism, devils, spirits, charms, absent treatment and many other forms are widely prevalent. They are applied for all purposes, from financial and social betterment to the healing of the sick and after-care of the soul. They are the elements used by the faker to rule an emotional, superstitious, ignorant, infantile-minded people for good or bad. The success attained is an indication of the psychologic condition of the masses.

THE SPINAL FORMS OF EPIDEMIC ENCEPHALITIS *

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The two outbreaks of epidemic encephalitis which occurred during the years 1918 and 1919 and the almost endemic condition which has continued during the year 1920 have served to provide us with material which has enriched our knowledge of the physiology and pathology of the central nervous system. Within the annals of medical history, we look in vain for records which can vie with the pages which have been written on neurologic symptomatology and pathology during the last three years. No system has been spared the ravages of this disease, and true to form, each system has stamped its signature on some one or other form of this most varied syndrome.

Prior to our more intimate and extensive knowledge of this disease, we had granted with but little or no discussion the dictum that syphilis was par excellence the master mummer of medical and neurologic science. We formerly believed that syphilis was the most protean of all infections involving the central nervous system, but epidemic encephalitis can lay claim to a teleologic influence over a wider range of symptoms, syndromes and morbid pictures than syphilis or any other disease with which we are familiar.

Perhaps one of the most valuable contributions to our knowledge of the nervous system, with which epidemic encephalitis has supplied us, is the conception that many of the diseases which formerly we judged to be due to chronic degenerative changes may now be definitely placed on an acute, subacute or chronic infective basis. How important and far reaching this new conception of many old familiar pictures may prove to be is difficult to say, but it is probable that the added zest and enthusiasm supplied by this freshening interest will result in a recrudescence of study of neurologic pathology.

The infective agent of epidemic encephalitis is preeminently one whose major affinity is for the cortex of the cerebral hemispheres and the substance of the brain stem. This characteristic was responsible

* Read before the Association for Research in Nervous and Mental Diseases, Dec. 29, 1920.

for the various names which were applied to the disease, and its more disseminate character has only disclosed itself through the study of combination and transition forms. Spinal forms were originally considered to be unusual forms of poliomyelitis associated with somnolence, and it was only after the number of cases became greatly multiplied and our attention was directed more closely to the disease that the widespread distribution of the infective agent was recognized. Many cases which proved puzzling and often not susceptible of classification have in retrospect been fitted into their proper diagnostic folder as sporadic cases of epidemic encephalitis.

The central nervous system outside the cerebral cortex and the brain stem presents a number of structures which may be the locus *resistentiae minoris* for the virus of the disease; the spinal meninges, the ventral and dorsal roots of the cord, the dorsal root ganglions and the spinal cord itself. All of these, or each one, may be the site of the development of the morbid process. Pure examples of infection of one or another of these structures are rare, and it is the rule to observe symptoms which are referable only to a combination invasion of these tissues. In each picture, however, will usually be found some predominating symptom which will indicate that the major part of the infection involves one or the other of these structures.

According to the figures collected by Dr. Israel S. Wechsler, out of 864 cases, forty-four were described as myoclonic, twenty-nine as radicular, neuritic or neuralgic, twenty-four as meningitic, sixteen as myelitic or spastic, five as paraplegic, two as ventral poliomyelitic and one as dorsal poliomyelitic, a total of 121 cases. It is impossible to state definitely how many of these cases are purely spinal, as that localization is considered in this paper, but at least twenty-three can be so judged, the myelitic, paraplegic and ventral poliomyelitic. How many of the myoclonic cases can be considered as spinal in origin is questionable and the number could be determined only by a close scrutiny of the individual records. As is often the case, the study of necropsy data only serves to obscure further the picture and render accurate differentiation and classification much more difficult or well-nigh impossible on account of the widespread involvement which is shown by a study of the pathologic material. Clinical data seem of relatively greater value in diagnosis than the pathologic findings. All forms seem to be combined forms, and only predominating symptoms determine the disease picture.

Although the dorsal root ganglions are to all intents and purposes but segregated parts of the gray matter of the cord and belong to the dorsal gray of the spinal cord, it will be taken for granted that the involvement of the dorsal root ganglions by the infectious agent of

epidemic encephalitis with the production of typical herpes zoster has been considered under a separate heading by the Association.

The types of epidemic encephalitis which are apparently mainly spinal in character may be divided into two groups: (1) the ventral poliomyelitic type of epidemic encephalitis, and (2) the transverse myelitic form of epidemic encephalitis.

The ventral poliomyelitic form of epidemic encephalitis may in turn be divided into two separate subgroups: (a) the irritative type, and (b) the paralytic type.

This division of the forms of the disease affecting the ventral gray matter of the spinal cord is largely symptomatic and is made in order that the manifestations of the disease may be separately considered. It is believed that the two forms merge one into the other and further that the paralytic type is often the end-result of the irritative type. It is clearly recognized that the irritative form may clear up at any time, and recovery may take place without any apparent permanent destruction of ventral horn cells and also that the paralytic form may develop full-blown without the picture being ushered in by the irritative phenomena. The second form is apparently very rare and is included with but little evidence on which to base its claim even to a precarious existence. It seems highly probable that such cases have occurred, and it is included as a type on largely theoretical grounds. As an end-result, cases of the paralytic form are being seen in increasing numbers and the majority of the irritative forms, either myoclonic or fibrillary, eventually present some lower motor neuron defect.

In considering the case reports as found in the literature, it has become increasingly clear that the point at which cortical irritation and spinal irritation should be differentiated is at times very difficult to determine. Under similar headings, cases have been reported whose manifestations pass from the coarse segment movements of a jacksonian type through all the gradations down to the fine fibrillations characteristic of ventral horn cell irritation.

The criterion for the differentiation between these various types of movement resulting from some irritative factor is the type of movement. It is clearly appreciated and understood, that movement resulting from irritation of the cortical level of the motor apparatus resembles volitional synergized movement. The cortex is motofacient, and stimulation of that part of the brain results in a definite movement in one direction or another. Such a movement partakes of a voluntary character. It is a synergized movement in which definite synergic groups participate and in which there is an integration of synergic units, evidencing the motor concept, formula and pattern and the collaboration of the cerebellum in the movement initiated by the cere-

bral cortex. These movements are the wellknown and recognized movements of jacksonian epilepsy resulting from cerebral irritation, either by neoplastic growth or inflammatory exudate. The functional tics and spasms also fall into this group. These movements have been recognized and studied for many years, and their characteristics are so well known that there is not a great deal of confusion or misunderstanding in regard to them.

It has been estimated that there are in the vicinity of 25,000 Betz cells in the precentral convolution and 250,000 ventral horn cells. Hence we conclude that each Betz cell controls a group of at least ten ventral horn cells. We also know that the Betz cells do not exist as solitary cells, but that they are arranged in groups of varying sizes. Any irritative lesion in the cortex would without much doubt be of sufficient extent to involve at least a group of such Betz cells if not several groups. The consequent result of such stimulation will be the contraction of not only a tenfold series of ventral horn cells, but also additional series dependent upon each added Betz cell affected. A still further amplification will result from the impulses which pass coincidentally from the stimulated cortical area to the cerebellum by way of the fronto-ponto-cerebellar tract, thus evoking the synergizing and coordinating function of the cerebellum. This clearly removes from the cortical level of irritation the localized and limited myoclonic contractions which may be seen playing over the surface of the muscle in the myoclonic form of the disease, producing no movement in the part, but only lightning-like, darting muscular contractions.

In a much less favorable state is our knowledge and appreciation of the movements resulting from the highest subcortical motor stratum, the corpus striatum. As a result of the flood of illumination which has of late been thrown on the functions of the basal ganglions by the French, English and American investigators, we have perhaps swung somewhat too far in our inclination to attribute all the unusual and bizarre types of involuntary movements to the influence and sphere of the corpus striatum. Nevertheless, it can scarcely be doubted that many, if not all, of the choreas, athetoses, spasms and dystonias can be laid with perfect propriety at the door of the corpus striatum. These movements are so well known that it is scarcely necessary to dwell upon them. This corpus striatum level of motion may be roughly characterized as the automatic associated stratum of movement, the phylogenetically conditioned and inherited type of movement.

The cerebellum, being definitely a nonmotofacient structure and responding to stimuli from levels above and below, and being incapable of independent irritability, has no distinctive level of motion, and we can ignore it in this connection.

The lowest level of motion except for direct muscular irritability with which we are not concerned, lies in the cells of the gray matter of the spinal cord and constitutes the spinal level of movement. These movements consist in contractions, at the maximum, of limited muscle groups and, at the minimum, single muscle fasciculi. The contractions of muscle groups are resultant upon the stimulation of a connector or association neuron by means of which a single impulse coming down from above is translated and enlarged into a contraction of a small group of muscles. These contractions are such as to produce essentially the same result, i. e., flexion or extension, abduction or adduction. There is no real synergism in these groups, there being no check and dominant element, but merely the cooperation of different muscles in producing a single movement, each contribution being essentially of the same type. It is evident that such contractions fall below the levels previously examined and do not partake of any voluntary or automatic associated character. They are simply purposeless irregular contractions, at most producing a simple movement in some direction and in lesser degrees causing the contraction of a portion of a muscle or single muscle fasciculi.

The coarse movements are usually characterized by irregular twitches which are exceedingly rapid and irregular in rate, rhythm and extent. These are called myoclonic contractions or myoclonus. They are probably dependent upon the stimulation of a small group of ventral horn cells which control a part of a muscle or a single small muscle.

At times, the type of contraction observed is quite different, a slow vermicular contraction commencing at the proximal end of the muscle and running down over the muscular belly to its distal extremity. This type of contraction probably corresponds to the stimulation of a connector or association neuron supplying groups and relays of ventral horn cells which are affected in varying degrees by the infection and whose rate of response to stimulation has been slowed down from the normal rapid reaction to the connector influence. As smaller and smaller groups of ventral horn cells are affected, the portion of the muscle belly responding to the nerve cell stimulation becomes smaller and smaller; a diminishing scale of contraction down to the typical contraction of almost a single muscle fasciculus. These are the typical fibrillary contractions with which we are so familiar and which are pathognomonic of ventral gray column cell irritation. Never producing a movement of the muscle as a whole, they can be seen producing a constant, unceasing ripple of motion over the surface of the muscle mass, often repeated in the same muscle fasciculus with lightning-like rapidity and then flitting to other fasciculi.

Another feature of many of the myoclonic types of the disease is the fact that the contractions have been preceded for a few days by lancinating pains which are typically radicular in character and distribution. The interval between the appearance of the pain and the incidence of the myoclonic contractions probably indicates the interval occupied by the passage of the infective agent from the roots of the spinal cord to the deeper parenchyma of that organ itself.

The irritative form may either persist for some time and then diminish in intensity and finally clear up with greater or less degree of disability or it may pass over into the paralytic form of the disease as a result of the ventral horn cells succumbing to the continued influence of the infective agent.

The paralytic form resembles closely that seen in anterior poliomyelitis or infantile paralysis when the volume of the infection at its onset is so overwhelming that the function of the ventral horn cell is abolished at the beginning of the disease. The paralyses are typically spinal in character, either a small group of muscles, one muscle, or a portion of one muscle being affected, there being usually no indication of a simultaneous involvement of synergic groups. This type is accompanied by all the manifestations of the destruction of the final common pathway, atonia, areflexia, atrophy and the abolition of all the various types of motor activity and response.

The transverse myelitic form of the disease resembles in all particulars, the symptom picture occasionally seen in infectious processes and in compression of the cord by fracture or neoplasm. The lesion may be complete or incomplete, depending upon the extent and severity of the process shutting off the body below the site of the lesion. The type of the syndrome in the partial transverse lesion will, of course, depend upon the anatomic structures involved. One interesting feature that has been seen in a number of such cases of the disease is the presence of a xanthochromia without the complete syndrome of Froin being present.

These transverse myelitic forms have been considered by some to be accidental occurrences in the course of an epidemic encephalitis and to be usually the result of a hemorrhage from a vessel weakened by the effects of the round cell infiltration and the perivascular rarefaction, or to be the result of an anemic or hemorrhagic infarction from thrombosis of some of the spinal vessels. This view may be upheld by the occasional occurrence of xanthochromia. It will be recalled that transverse myelitis is practically unknown as a form of poliomyelitis, and since the lesions of the two diseases are so similar, this may lend some confirmation to the views heretofore expressed.

REPORT OF CASES

The case herewith reported from the service of Dr. Foster Kennedy at the Neurological Institute, New York, is an example of the irritative form of the disease with myoclonic contractions.

CASE 1.—History.—G. M., aged 19, born in the United States, complained of symptoms the onset of which had occurred seven weeks before, with pain and soreness in the head, which in a few days spread to all the extremities and the thorax. A few days later, retention of urine developed. There were insomnia, a transient diplopia and a tremor with twitching movements. The patient had had the usual diseases of childhood. The family history was irrelevant.

Physical Examination.—The patient was confined to bed and seemed to prefer to sit up. There was some bending forward of the head and a definite fixity of facial expression.

There was a coarse tremor of the upper and lower extremities and a transient cog-wheel phenomenon on passive flexion of the forearm. There were quick, brief, jerky movements of the toes and feet and myoclonic contractions of the dorsal muscle groups in the thigh. There were definite rhythmical twitching movements of the abdominal walls which were somewhat more appreciable to palpation than to inspection. There was some slight voluntary control of the movements.

Reflexes: Knee jerks were both sluggish; ankle jerks were very sluggish, and abdominal reflexes were absent. No pathologic reflexes were present.

Sensibility: No definite changes in sensibility were made out; but there seemed to be a slight diminution in touch and pain below the middle of the sternum.

Cranial Nerves—Ocular Movements: There was some limitation of downward movement of both eyes and a slight external rectus paresis on the right side. The pupils were dilated and sluggish to light and accommodation. The remaining cranial nerves were normal.

Spinal Fluid: Perfectly clear spinal fluid was found under moderately increased pressure. No globulin was found; Fehling's solution was reduced; the lymphocyte count was 5; the Wassermann reaction was negative, and the colloidal gold reaction, 011100000.

Blood: This examination revealed: hemoglobin, 76 per cent.; erythrocytes, 4,200,000; leukocytes, 12,750; polymorphonuclears, 65 per cent.; lymphocytes, 32 per cent.; basophils, 1 per cent.; transitionals, 1 per cent., and eosinophils, 1.

Urine: On examination the urine was found to be yellow, turbid and acid. The specific gravity was 1.018. It contained a marked trace of albumin, an excess of indican, epithelial and squamous cells.

The subjoined case is an example of the myoclonic-fibrillary type followed by the paralytic phenomena.

CASE 2.—History.—A. M., aged 32, born in the United States, metallurgist, complained chiefly of weakness in the left side of the abdomen and the left leg. He gave a history of typhoid six years before; quinsy, chicken-pox, measles, mumps, all in childhood; gonorrhea two years before, and exposure to syphilis. He drank five cups of tea each day, one cup of coffee each day, used a moderate amount of tobacco, but no alcohol. He slept well. His appetite was good, he used liquid petrolatum to keep the intestinal condition normal.

Urination presented no abnormalities. There was no history of accident, operation or injury. He had had no convulsions, dysphagia, dyspraxia, no visual or auditory disturbances, no headache or vomiting. He had been married nine years and his wife had never been pregnant. The present symptoms began in April, 1920, with a feeling of "pins and needles" followed by pain in the left instep, which became much worse, until he could no longer place his foot on the floor without great pain. The pain was at first localized, very sharp and stabbing in character. In a short time the pain became shooting and lancinating and spread to the right leg. It was worse in the left leg. These pains lasted for about ten days and then gradually subsided. Before they left entirely, twitching movements appeared in both calves and spread to both thighs and the abdomen. These movements have persisted up to the present time although they have decreased in severity and extent.

REFLEXES ELICITED

Deep Tendon Reflexes	Left	Right
Jaw	2	2
Pectoral	1	1
Biceps	2	2
Triceps	2	2
Radial	2	2
Ulnar	2	2
Wrist	2	2
Patellar ..	2	1
Achilles	0	0
Cutaneous Reflexes		
Ciliospinal	2	2
Supra-umbilical	?	2
Suprapubic	0	2
Upper lateral abdominal	?	2
Lower lateral abdominal	0	2
Cremasteric	?	?
Babinski, etc.	0	0

The patient has no recollection of the period following the onset of symptoms for he was delirious, actively hallucinating and showed some expansive tendencies. At that time, due to the physical examination, contradictory serologic reactions and the history of an exposure to syphilis, he was treated for that condition with a reported marked amelioration of the symptoms. A report from the hospital at the time of his admission states that he presented these symptoms: insomnia, restlessness, a delayed reaction to light and accommodation, and the mental state mentioned. A neurologic report emphasizes the pupillary condition, inequality of the reflexes, the myoclonic contractions, a diplopia and a possible left facial weakness with a diagnosis of epidemic encephalitis. There was considerable sensory disturbance on both sides below the waist, involving all types of sensibility. There was considerable atrophy. The patient was discharged from the hospital early in September and has improved steadily ever since.

Physical Examination.—Dec. 30, 1920, the physical examination revealed bilateral flatfoot with some eversion. He could not walk on the toes or heels. The left foot was weaker than the right. There was some steppage, the feet and legs rotated internally in walking. There were no equilibratory distur-

bances in walking. Coordination was normal in all respects. There was no adiadokokinesis or dysmetria. Skilled acts were entirely normal.

The muscles of the abdominal walls, especially the recti of both ventral and dorsal femoral regions and ventral and dorsal tibial regions, presented a marked state of involuntary activity. This muscular activity was markedly increased by stretching the muscles. The contractions could be regulated by conscious effort only to a very slight extent. The contractions were both myoclonic and fibrillary and seemed to affect equally all of the muscles mentioned. There was a continual rippling fibrillary play of contractions which were at times superseded by lightning-like shooting contractions of larger parts of the muscles affected.

The reflexes that were elicited are given in the accompanying tabulation.

Muscle strength was diminished in the abductors of the thighs of both sides; the adductor of the left thigh was affected more than the right. The left leg was most affected. The movements of the foot were more affected than those of the leg. Power in the ventral and dorsal tibial groups was very much affected, dorsal flexion being affected more than plantar flexion.

There was a definite atrophy of the left thigh and of both legs, also of the left gluteal region: thigh, left, 38.5, right, 42.5; calf, left, 27, right, 28.

The electrical reactions were very sluggish. Faradic contractility was maintained and there was no reversal of the galvanic formula. There were no abnormal associated movements. Tactile sense was reduced below the second lumbar segment and almost abolished over the first to fourth sacral segments on both sides, more marked on left than right. There was analgesia over the fourth lumbar to the fifth sacral segment on the right, and over the second lumbar to the fifth sacral segment on the left.

The temperature area was the same as for pain. Vibratory sense was normal.

Muscle-tendon sense was normal. There was some itching over the second to fifth sacral segments on both sides, and scratching had produced some trophic ulcers on the dorsum of the right foot.

The first and second cranial nerves were not tested.

Examination of the oculomotor apparatus revealed: pupils, right, 3.25 mm.; left, 3.00 mm.; round, regular and central; normal reaction to light and accommodation; no hippus; normal position; no voluntary limitations; no nystagmus; no abnormalities of iris or media. The remaining nerves were normal.

The thoracic and abdominal viscera were normal. The pulse was 84, full and regular. Blood pressure was 125 systolic and 95 diastolic.

The following case is an example of the transverse myelitic type of the disease from the service of Dr. Frederick Tilney and Dr. Hubert S. Howe, at the Presbyterian Hospital, New York.

CASE 3.—History.—S. F., aged 50, Jewish, whose family and previous history was irrelevant, for the last five weeks had suffered from dizziness, diplopia, headache and paralysis of the left side of the face. There had been a definite general weakness, both eyelids drooping, and for the last two weeks, the right eye had been closed and swollen. There had been some deterioration in the sight of the right eye. The patient could not raise her legs. She suffered from nocturia. The menopause had occurred fourteen years before.

Physical Examination.—There was some enlargement of the heart in both directions. A faint blow was heard over the apex. Blood pressure was 190 systolic and 108 diastolic. There were some varicosities in both legs, and some edema.

Neurologic Examination.—The attitudes were as described. There was no thoracic respiration; the right eye was closed completely, the left partially; the left side of the face was paretic, the legs were slightly abducted, externally rotated and flaccid. The patient could not walk. Coordination was normal in the right arm. There was some swaying in the left arm. The lower extremities were not tested. There were no cerebellar disturbances. Reflexes of the upper extremity were: right, 2, left, 2; lower extremity, right, 0, left, 0; abdominal reflexes, right, 0, left, 0; pathologic reflexes, right, 0, left, 0.

There were no abnormal associated movements.

The patient could not raise her head or sit up. The movements of the upper extremities were weak and slow. There was no movement of the lower extremities except slight flexion and extension of the toes.

Tone was much reduced in the lower extremities.

There was a band of anesthesia on the left chest corresponding to the second to fourth thoracic segments and a similar area over the fourth to seventh thoracic dermatomes on the right side of the thorax. There was anesthesia over the second and third sacral segments in both legs. These changes involved all types of sensation.

There was slight papillitis in each eye. The pupils were equal, regular and central. The right reacted sluggishly to light; the left was slightly more sluggish. The right eye was closed; the left was almost closed. The right eye was immobile; the left eye showed a deficient internal rectus. There was a partial left seventh. The other nerves were normal.

Lumbar puncture revealed a perfectly clear, straw-colored fluid under increased pressure, which contained 32 cells and a few erythrocytes. The globulin was ++. The Wassermann reaction was negative.

The urine was amber and contained a heavy trace of albumin and a few casts.

Blood examination revealed: hemoglobin, 75 per cent.; erythrocytes, 3,500,000; leukocytes, 8,600; polymorphonuclears, 75 per cent.; lymphocytes, 25 per cent.

As a result of lumbar puncture, the picture became that of a total transverse lesion at the upper level indicated in the physical examination, that is, at the second thoracic dermatome. The paraplegia became complete, with retention of urine and feces. The patient subsequently developed a bronchopneumonia and died. No necropsy was permitted.

CONCERNING THE CLINICAL CLASSIFICATION OF INTRACRANIAL TUMORS *

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In the course of an address (unpublished) before the American College of Surgeons,¹ on "Brain Tumor Statistics," it was pointed out by Dr. Harvey Cushing that there was great difficulty in comparing statistics from different clinics because of the great variation in classification. It is believed that a record of the method employed in a clinic where large numbers of such cases are handled will be of value. The routine series entering the Peter Bent Brigham Hospital between April 1, 1919, and Dec. 1, 1919, has been chosen, all these cases having been under my personal supervision.

There were admitted between the dates given, 118 patients supposed to be suffering from some sort of intracranial new growth involving the brain. Some of the difficulties involved in classification may be appreciated from the following history which will be, in common with all the succeeding ones, condensed to positive findings, omitting the results of all other routine examinations unless they have a bearing on the case.

CASE 1 (P.B.B.H. Surg. No. 10598).—*Endothelioma of the right occipital lobe. Preliminary subtemporal decompression. Subsequent extirpation in a two-stage operation. Recovery.*

June 7, 1919, M. W., aged 61, a tailor, recommended by Dr. Nightingale of Worcester, Mass., entered the hospital complaining of failing vision and weakness of the right leg.

Chronology of Symptoms.—For six months he had suffered with occasional frontal headaches, not severe or continuous, and progressive failure of vision; for three months dull aching pain in the right arm and leg at times. Lately there had been weakness of the right leg.

Positive Findings.—There were tortuosity of the retinal vessels with patches of exudate along their course, no measured elevation; slight asymmetry of surface temperature, the left arm being warmer than the right; slight motor weakness in the left leg.

Discharge Diagnosis.—Brain tumor and arteriosclerosis were suspected. He was admitted for the second time on June 23, 1919.

* From the clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital Boston.

1. New York City, Oct. 21, 1919.

Course.—Since he had been discharged the headaches and pains in the right leg and arm had entirely disappeared.

Positive Findings.—Inconstant left ankle clonus, marked dilatation of extracranial blood vessels and bilateral low-grade choked disk were present.

Clinical Diagnosis.—Possible endothelioma.

Operation.—June 27, 1919, a right subtemporal decompression was performed. The brain was tense and vascular.

Postoperative Notes.—Ankle clonus disappeared. The decompression area was tense. He was discharged July 9, 1919.

Discharge Diagnosis.—The diagnosis was tumor cerebri, unverified; possible endothelioma.²

The patient was admitted for the third time on Sept. 3, 1919.

Course.—He had continued free from headaches. Vision was slightly improved. For six weeks there had been a feeling of numbness and tingling in the left arm and gradual development of weakness.

Positive Findings.—The decompression area was tense and bulging. The extracranial veins were enormously dilated. There was bilateral low-grade choked disk with secondary optic atrophy. The left pupil was slightly larger than the right. There were left homonymous hemianopsia; slight emotional left facial weakness; hypesthesia over the entire left half of the body below the trigeminal area, more evident toward the distal portions of the extremities. Sense of position was impaired in the left arm and leg. The tendon reflexes were exaggerated in the left arm and leg, with ankle clonus.

Clinical Diagnosis.—The diagnosis was cerebral tumor of the right post-central region.

Operation.—Sept. 5, 1919, an osteoplastic exposure of the posterior right hemisphere was made. Palpation of the dura disclosed a hard area in the posterior extremity of the field, suggesting underlying endothelioma. Closure was made without opening the dura because of vicious bleeding from the bone.

Second Stage Operation.—Sept. 12, 1919. The bone-flap was again reflected. An occipital lobe endothelioma was extirpated from the attached surface of the falx. Blood transfusion was performed.

Postoperative Notes.—Recovery was rapid and uneventful in spite of the fact that the patient insisted on fasting during the Jewish holidays. He was discharged Sept. 30, 1919.

Discharge Diagnosis.—The diagnosis was cerebral tumor, verified; endothelioma.

Comment.—Although at the time of his first discharge, because of the indefinite nature of his complaints and in view of his age, it was felt that arteriosclerotic changes would account for his troubles, the possibility of tumor was not overlooked, and he was told to report occasionally for observation. It will be noted that at the time of his second admission the only motor sign was on the left side, while all his symptoms had been on the right. A right subtemporal decompression was done to relieve his eyesight while more definite localizing

2. Dr. Cushing based this impression solely on the dilated vessels of the scalp. The roentgen-ray findings were negative. He felt so sure that he sent for the patient to return although the old man was well satisfied with the results of his decompression.

signs developed. His third admission resulted from a letter to his physician inquiring about his progress. A glance at the following classification will show that this patient on his various admissions was successively in each of the three main groups in which tumor cases have come to be tabulated in this clinic.

CLASSIFICATION OF AUTHORS' CASES

The 118 cases were divided thus:

1. Brain tumor suspects	31
A. Clinically nontumor	23
Cases admitted as brain tumor which after thorough study were not so regarded.	
B. Clinically doubtful	5
Cases admitted as brain tumor in which the findings were doubtful but operation was performed and no evidence of tumor found.	
C. Clinically tumor	1
Clinically localizable tumor, but operation although exposing location disclosed no evidence of tumor.	
D. Verified nontumor	2
Clinically localizable tumor, but pathologic lesion determined and absence of tumor verified at necropsy.	
2. Brain tumor, unverified	24
A. Unoperated	13
B. Unlocalized	5
Decompressed to relieve pressure symptoms.	
C. Undisclosed	6
Explored but no lesion found.	
3. Brain tumor, verified	63
A. At operation	57
B. At necropsy, unoperated	1
C. At operation and necropsy	1
D. At necropsy, unverified at operation	4

The diagnosis written on the history sheet at the time of discharge of the patient usually includes only the main group plus the presumptive or established pathologic diagnosis, but all the necessary information for placing a case in the foregoing scheme may be gathered from the front sheet of the history after discharge. For example, if the diagnosis reads "brain tumor suspect; encephalitis" and there is no record of operation or necropsy examination, the patient obviously belongs in 1 A; if the necropsy findings are recorded, the case falls in 1 D.

The patient whose history was given at the outset, on his first admission would have been placed in Group 1A as tumor suspect, clinically arteriosclerosis. On his second admission, owing to his choked disk and the tension found at operation, his case fell into 2 B or tumor unverified, though operated, because unlocalizable and therefore

decompressed. At the last admission he was shifted to Group 3 A when the tumor was verified at operation.

Many times the final classification was determined by developments during the patient's stay in the hospital. The clinical picture was often very doubtful but gave sufficient grounds for undertaking an exploratory operation. If, during the course of the operation, no evidence of tumor was disclosed, the patient was classified under 1 B; if evidence was disclosed, (excessive tension, etc.) under 2 C; if the tumor was found and verified, under 3 A. The necessity for change sometimes occurred after a patient had left the hospital, as happened with L. McN., Surg. No. 11187, reported elsewhere,³ who was discharged as "brain tumor unverified, presumably basilar glioma" and later came to autopsy. Examination of the brain made it necessary to shift her from 2 C to 1 D. There was found a meningo-encephalitis.

I. BRAIN TUMOR SUSPECTS: THIRTY-ONE CASES, 26.27.

PER CENT.

A great many patients were admitted to the clinic with a diagnosis of brain tumor who proved to have some other disease. They have all been placed in this main group, which will be examined in some detail as at once the most difficult and most interesting. A straightforward case of acoustic neuroma or pituitary adenoma, easily recognized and routinely removed, causes little excitement, but a borderline case in which one must employ all his wits and every instrument of precision at his command arouses the most intense interest.

A. Clinically Nontumor.—In this group are placed patients admitted with a diagnosis of presumptive brain tumor in whom, on examination, conditions other than tumor were found, which it was felt would explain the symptoms. No operation was therefore undertaken. They were usually referred by general practitioners, although occasionally by neurologists.

The clinic has of course, little control over the number of cases that fall into this group. When a physician writes that he has a case of brain tumor which he wishes to refer there is little possibility of determining that no tumor is present before the arrival of the patient. And often these patients have puzzling and interesting conditions. The following case of unusual interest is a good example.

CASE 2 (P.B.B.H. Surg. No. 10229).—*Intracranial arteriovenous aneurysm with tumor syndrome of the cerebello-pontile angle. Dislocation of atlas.*

3. Bailey, P.: Contribution to the Histopathology of "Pseudotumor Cerebri," Arch. Neurol. & Psychiat. 4:401, 1920.

April 2, 1919, Francis T. N., aged 48, a miner, was admitted to the hospital, referred by Dr. A. F. Fischer of Hancock, Mich., with complaint of pain in the back of the neck, dizziness, double vision and facial paralysis.

Chronology of Symptoms.—August, 1914, a singing noise in the left ear began which continued for two years, then ceased, leaving partial deafness. November, 1916, dizziness developed. April, 1917, diplopia appeared. December, 1917, paralysis of the left half of the tongue and a sensation of numbness and thickness appeared. February, 1918, there was weakness of the left side of the face. October, 1918, there was complete paralysis of the left side of face. June, 1918, severe pain in the neck followed a misstep. January, 1919, the left ear drum was punctured; this was followed by repeated severe hemorrhages. February, 1919, there was severe pain in the left ear followed by discharge.

Positive Findings.—These were: left pupil larger than the right; paralysis of the left external rectus; fine nystagmus on looking to the left; hypersensitivity to pain in the area of the middle division of the left trigeminus; left peripheral facial paralysis; disturbance of taste on the posterior part of the tongue; left recurrent laryngeal paralysis; Rinne's test negative in the left ear; atrophy of the left half of the tongue; slight ataxia and adiadokokinesis in the left arm; pain on flexing the head on the chest; red mass projecting into the left external meatus; shrill and continuous humming murmur over the left carotid sheath, heard best around the left ear. In the roentgenogram there was seen to be a dislocation of the atlas.

Discharge Diagnosis.—Brain tumor was suspected clinically; there were intracranial arteriovenous aneurysm and dislocation of the atlas.

Comment.—Most cases in this group are not of sufficient interest to be detailed here. They will be reported elsewhere. It might, however, be of interest to note the final diagnosis arrived at in this clinic. There were twenty-three cases, distributed as follows: Ménière's syndrome, essential epilepsy, cerebral thrombosis (with epilepsy), encephalitis (lethargic), polyneuritis (with encephalitis), cerebral hemorrhage, congenital malformation, marasmus (with optic atrophy), hysteria (simulating pituitary tumor), otosclerosis, post-traumatic psychosis, congenital nystagmus, hysteria (simulating acoustic tumor), syphilitic meningitis, otitis media, cephalalgia (neurasthenic), senile psychosis, intracranial arteriovenous aneurysm, choroiditis, psychasthenia, cerebral embolism, toxic amblyopia and multiple sclerosis. Occasionally such a patient returned, as already noted in the case of M. W., Surg. No. 10598, from whom an endothelioma was removed from the falx cerebri in the occipital region on his third admission.

B. Clinically Doubtful.—In all the cases in the preceding group it was reasonably certain that a tumor was not present, but occasionally a patient would present himself with sufficient evidence to lead to an exploration, although the diagnosis was questionable. Often in such cases a tumor was found. If, however, normal brain or definite pathology other than tumor was disclosed, the patient was placed among the suspects. The absence of tumor of course cannot always be finally

established without a necropsy examination, even though other lesions are found. An example will be given later (F. S., Med. No. 11985).

Five instances of this sort were found, and each presents features of interest.⁴

CASE 3 (P.B.B.H. Surg. No. 11271).—*Epilepsy with mental deterioration and doubtful symptoms. Exploration. Negative findings.*

Oct. 6, 1919, John W., aged 25, a farmer, recommended by Dr. E. A. Ludden of North Brookfield, Mass., was admitted to the hospital with complaint of "fainting spells."

Chronology of Symptoms.—For nine years he had had attacks of transitory loss of consciousness, often, but not always, accompanied by muscular twitchings. According to the mother, the right side was always involved more than the left and at times only the right side was involved. There were no involuntary twitchings. The attacks occurred with increasing frequency until lately practically every day brought an attack. For four months he had had hallucinations of sight. Lately he had practiced sodomy and other sexual perversions.

Positive Findings.—The temperature was markedly irregular ranging from 97 to 101 F. The tendon reflexes in the lower extremities were sluggish. The roentgenograms showed enormous dilation of the diploic veins with pressure absorption in the frontal region. There was marked mental retardation. The patient was seen to have a typical jacksonian attack beginning in the right arm and extending to the face, while standing, without falling but with transitory unconsciousness. The perimetric examination was questionable because of lack of cooperation.

Clinical Diagnosis.—There was a possible endothelioma of the left frontal region.

Operation.—Nov. 1, 1919, an osteoplastic exposure of the left frontal lobe was made. The extracranial vessels were much dilated, and there was excessive bleeding from the large diploic veins. The dura was tense, and at the upper anterior corner was a thickened and roughened area. Underneath no tumor was found, although the brain was congested and tense. An attempt to enter the ventricles was unsuccessful.

Postoperative Notes.—Recovery was uneventful, and the patient was discharged Dec. 18, 1919, unimproved.

Discharge Diagnosis.—Brain tumor was suspected; epilepsy of doubtful etiology.

Comment.—The history in this case was not reliable. Neither the patient nor his mother could give a connected story. Objective signs of destruction of motor cells were lacking, but the attack seen was quite definitely jacksonian. Pressure signs were absent, but the dilated extracranial vessels and enormous diploic veins were suggestive. During the operation, the dilated veins, the bleeding diploe, the absorption of bone, the discolored dura, everything down to the actual moment of opening the dura seemed to increase the certainty of the diagnosis.

4. The publication of this article was delayed by the absence of the writer from the clinic for several months. This section is left as originally written, and the progress of the patients since discharge is indicated in the footnotes.

The nature of the lesion is uncertain, and the gradually increasing mental deterioration, the epileptic attacks gradually becoming more frequent, the circulatory changes in the skull and the irregular temperature require some explanation. A deep lying glioma may yet be found.⁵

CASE 4 (P.B.B.H. Surg. No. 11130).—*Convulsions of cerebellar type with doubtful tumor symptoms. Exploration. Negative findings.*

Sept. 9, 1919, Eloise T., aged 44, a housewife, recommended by Dr. J. W. J. Marion, of Calais, Me., was admitted to the hospital with complaint of headaches and vomiting.

Chronology of Symptoms.—For six months she had had attacks of severe suboccipital headache with vomiting, unaccompanied by nausea. The attacks were sudden in onset and short in duration. Lately they had been much more frequent. For the past week there had been slight weakness and tremor of the right hand and prickly sensations in both arms.

Positive Findings.—There were fine nystagmoid twitchings on lateral movements of the eyeballs; slight incoordination of the left hand; tendon reflexes on the left side slightly exaggerated; slight edema of the nasal borders of the optic disks.

Clinical Diagnosis.—The diagnosis was: possible cerebellar tumor.

Operation.—A bilateral suboccipital craniotomy was performed Sept. 15, 1919. There was no unusual tension, but considerable respiratory difficulty; no internal hydrocephalus. Puncture of each cerebellar hemisphere disclosed nothing abnormal.

Postoperative Notes.—During the first week there were periods when the respiratory rate reached as low as 7 per minute. The patient complained of pain in the arms at first but improved rapidly and was discharged Oct. 21, 1919, entirely free from symptoms.

Discharge Diagnosis.—Brain tumor was suspected; essential pathology undetermined.

Comment.—This patient lived in the Maine woods, far away from surgical aid, and since it was feared that a severe attack might prove fatal, in case a tumor were present, before she could be transported to a hospital, an exploratory operation was advised. If, in this case, an internal hydrocephalus had been disclosed, she would have been listed as having a case of cerebellar tumor, unverified. It may be that the

5. Note from the Monson State Hospital, Palmer, Mass., Oct. 20, 1920: "This patient died May 23, 1920, and the cause of death was given as epilepsy with status epilepticus. He was admitted to this institution Dec. 18, 1919. . . . His seizures averaged from three to twelve of the G. M. type and from none to 260 of the P. M. type. . . . I regret that no autopsy was obtained"

In this clinic a man was recently operated on, John P., Surg. No. 13161, who gave a history of mild sensory attacks over a period of sixteen years. Pressure symptoms and motor attacks had appeared only within the last six months. At operation an extensive cerebral glioma was found.

obstruction was too recent and transient to result as yet in a dilatation of the ventricles.⁶

CASE 5 (P.B.B.H. Surg. No. 11256).—*Internal hydrocephalus with post-operative cerebral hernia and cerebrospinal fluid leak. Successful closure.*

Oct. 2, 1919, Hayman C. H., aged 22, a bank teller, referred by Dr. J. W. Alsobrook, of Plant City, Fla., was admitted to the hospital with complaint of paralysis of the left arm and leg and discharge from the right side of the head.

Chronology of Symptoms.—December, 1918, he had an acute tonsillitis, followed by right otitis media. The ear drum was punctured, followed by persistent discharge. March 15, 1919, the discharge from the ear ceased. April 7, he had a headache. April 8, he had a violent headache and staggering gait. April 11, the ophthalmologist diagnosed choked disk and advised operation at once. Operation was refused. April 15, operation was performed. A transverse incision through the right temporal muscle was made. The temporal lobe was explored but no abscess was found. The wound was packed with gauze. April 17, another unsuccessful attempt was made to find the abscess. The wound was again packed and the packing was changed every day. Hernia of the brain developed. April 24, there was beginning weakness in the left arm followed soon by weakness in the left leg. May 1, the fungus sloughed entirely and the brain receded into incision. May 3, the incision had healed. May 5, the patient had severe headache. May 7, the wound was opened and kept open by drains ever since.

Positive Findings.—The findings were: a right temporal protrusion from which cerebrospinal fluid was escaping; bilateral secondary optic atrophy; fine nystagmus to right; left facial paresis; Weber's test lateralized to the right and Rinne's test negative for the right ear; deviation of the tongue to the left; paresis of the left trapezius; suboccipital tenderness on the right side; flaccid paralysis of the left arm except for some spasticity of the biceps and flaccid paresis of the left leg; exaggeration of biceps, triceps, periosteal, knee and ankle reflexes on the left side with a positive Babinski reflex and wrist and ankle clonus; hypesthesia of the left half of the body for touch, temperature and muscle sense; epigastric, abdominal and cremaster reflexes absent on the left side.

Clinical Diagnosis.—The diagnosis was possible cerebral tumor.

Operation.—Oct. 9, 1919, a right osteoplastic exploration disclosed a sinus leading down to an enormously dilated right ventricle. No other lesion was found. The defect was closed.

6. The patient was readmitted March 8, 1920, Surg. No. 12044. Vision had progressively failed to almost total blindness. Frequent attacks of vomiting developed, with generalized convulsions and incontinence. There were no headaches. Examination showed anosmia, bilateral primary optic atrophy, fibrillary twitching of the muscles around the mouth, absent knee and ankle jerks, slight ataxia of the hands and paresis of the left internal rectus muscle. March 19, 1920, a transfrontal operation revealed no pathologic changes in the region of the pituitary. The Wassermann test of fluid withdrawn from the lateral ventricle during operation was positive. All previous tests for syphilis had been negative.

A note from the husband, Nov. 1, 1920, stated: "Has been confined to her bed since (return from hospital); appetite exceptionally good and sleeps well but is gradually wasting away."

Postoperative Notes.—The defect remained closed. The patient was discharged Nov. 1, 1919, with the neurologic condition unchanged.

Discharge Diagnosis.—Brain tumor was suspected; there was internal hydrocephalus of unknown etiology.

Comment.—The surgeon first became suspicious of tumor when the wound would not close without development of pressure symptoms. There was evidence to support such a suspicion. In the first place, no abscess was found, and the subsequent course of the disease argued against the presence of abscess. Furthermore it was early for the development of abscess. Also the surgical damage, bad as it was, could hardly have involved any portion but the temporal lobe, while progressive paralysis of the arm and leg developed.

It is stated that at the time of the first operation a cyst was opened and clear fluid escaped. It seems most probable that the surgeon made an opening into the lateral ventricle and continued to keep it open by packing until the foramen of Monro became occluded. When an attempt was made to close the exterior opening, a unilateral hydrocephalus developed, giving rise to headache, which was relieved by establishing a permanent sinus. After the sinus was finally closed here, considerable pressure developed for a day or two and disappeared, which we may perhaps explain by supposing that the foramen was again forced open, or even that an opening was forced through the septum pellucidum, as often happens in internal hydrocephalus.

Why the abscess was searched for in the temporal lobe is not certain. The staggering gait would indicate the cerebellum as the seat of trouble. If an abscess were present in the cerebellum, draining the lateral ventricle would relieve the pressure symptoms, but after such a long interval, closure of the drainage sinus would surely have led immediately to a recurrence of the symptoms, for although a brain abscess may heal spontaneously, it rarely does so.

There is even some question as to the condition of the eyegrounds prior to the first operation. The information at hand states that there was present bilateral choked disk with destruction of more than half of each retina. It seems most likely that the patient had an acute labyrinthitis, and that all his later troubles were due to operative trauma. The paralysis was probably caused by disturbance of circulation in the area of the middle cerebral artery when the hernia sloughed.⁷

7. Note from physician, Oct. 14, 1920, states: "Since his return from the hospital he has had several attacks of distention of the cyst which required drainage. He is now able to walk with a cane and move his left arm slightly. His eyesight is not improved in the least. . . . His general health is good."

CASE 6 (P.B.B.H. Surg. No. 10355).—*Chronic circumscribed arachnoiditis with cerebellar tumor syndrome. Exploration. Death.*

April 24, 1919, Joseph E., aged 33, a cigarmaker, referred from the outpatient department, was admitted with complaint of occipital headache and staggering gait.

Chronology of Symptoms.—March, 1912, the patient developed attacks of severe headache, usually suboccipital. The attacks increased in severity and were always accompanied by vomiting and dizziness. Meanwhile diplopia developed, and his eyesight began to fail. August, 1912, a right subtemporal decompression was made at the Massachusetts General Hospital which disclosed a tense brain but no other lesion. Following the operation there was rapid improvement. Headaches, vomiting, diplopia and dizziness ceased. August, 1915, the patient noticed an increasing clumsiness of the right hand so that he was obliged to discontinue work. August, 1918, attacks returned of severe suboccipital headache accompanied by stiffness of the neck and great unsteadiness of the gait. The attacks were increasing in frequency and severity.

Positive Findings.—These were: marked bilateral suboccipital tenderness and stiffness of the neck; staggering gait with greater tendency to fall to the right; diminution of muscle tone and power on right side, with incoordination, ataxia and adiadokokinesis; rotatory nystagmus downward and to the left on looking upward; secondary optic atrophy; cranial defect under right temporal muscle, not tense nor bulging; and an astereognosis of the right hand with some lowering of two-point discrimination. In short, there were definite signs of cerebellar involvement, but at the same time an unmistakable sign of involvement of the left parietal region of the cerebrum.

Clinical Diagnosis.—The case was diagnosed as a possible double meningeal endothelioma.

Operation.—The usual bilateral exposure was made May 14, 1919. The arachnoid was thickened and adherent to the dura, and in it were huge cystic spaces. The cerebellum was much deformed. The left hemisphere seemed about one-half its normal size, and the right was almost totally destroyed, only the great fiber bundles remaining. On attempting to puncture the left ventricle much fluid was secured immediately under the dura so that there was undoubtedly here also an arachnoid cyst.

Postoperative Notes.—The patient recovered consciousness completely and the condition seemed satisfactory except for profuse sweating and hyperthermia until he died suddenly of respiratory failure thirty-six hours after the operation. Necropsy examination was refused.

Discharge Diagnosis.—Brain tumor was suspected; chronic circumscribed arachnoiditis.

Comment.—The presence of tumor was doubtful, without assuming the remote possibility of a double lesion. Of course, supratentorial tumors may give rise to slight cerebellar symptoms, but in this case both the cerebellar symptoms and the astereognosis were pronounced. Fearing a bad cerebellar seizure might prove fatal, an exploratory operation was considered justified.

CASE 7 (P.B.B.H. Surg. No. 10331).—*Hysteria with cerebellar tumor syndrome. Exploration. Negative findings. Recovery.*

April 20, 1919, Charlotte R. K., aged 33, a school teacher, recommended by Dr. Joseph Stanton of Boston, Mass., was admitted to the hospital with complaint of headache, vomiting, dizziness, double vision and attacks of unconsciousness.

Chronology of Symptoms.—At 4 years of age the patient had had an otitis media following scarlet fever. In 1913, she had a ventral suspension of the uterus performed. In 1915, some friends persuaded her to have her ear operated on although, except for a slight discharge, it caused her no trouble. In July, 1915, a radical mastoid operation was performed on her left ear. There was some delay in healing requiring skin graft, but final healing occurred in December, 1915. January, 1916, she suffered from nausea, double vision and severe dizziness. The symptoms subsided in ten days, but slight dizziness continued until summer. October, 1916, she had a similar attack accompanied by unconsciousness for three days at the onset. There was residual slight dizziness. January, 1918, she had a third attack accompanied by unconsciousness. Operation was performed through the external acoustic meatus. March, 1919, she had a fourth attack accompanied by unconsciousness. The old mastoid wound opened. Since that time she had remained in bed, never free from dizziness, nausea and double vision. She had had two other attacks of unconsciousness, one late in March and another just previous to admission.

Positive Findings.—These were: slight tortuosity of the veins of the retinae with obscuration of the nasal margins of the disks; paresis of the left facial nerve, apparently peripheral in type; Weber's test lateralized to the left ear; Rinne's test negative and auditory acuity much diminished in same; muscle tone markedly diminished on left side; Romberg sign positive; gait reeling with tendency to go to the left; slight incoordination of the left upper and lower extremities.

April 23, 1919, the patient was found in a strange semisomnolent state, with deep, rapid respirations, 24 per minute with expiratory grunt. She opened her eyes but did not respond to questions. The accessory respiratory muscles were called into play. The pupils were widely dilated. The veins of the fundi were dilated and tortuous, with edema of the nasal margins of the optic disks. There was no stiffness of the neck and no suboccipital tenderness. The rectal temperature was 99.2 F. The leukocyte count was 16,400.

Clinical Diagnosis.—This was possibly a case of extracerebellar tumor; there was probably a chronic arachnoid cyst of the left cerebellopontile angle. Consultation with Dr. Eugene Crockett, who concurred in this opinion.

Operation.—May 3, 1919, the usual suboccipital exposure was made. Although exploration of the left recess was carried down to the temporal bone and the seventh and eighth nerves were seen, no abnormality was disclosed.

Postoperative Notes.—The patient was extremely unruly during convalescence, screaming and crying almost constantly at first. She had one of her semisomnolent attacks, but was found at the same time to have secreted candy in her room, of which she consumed quantities. Suspicion growing that her troubles were mainly functional, she was given a vigorous lecture and became much more tractable. She was discharged June 7, 1919, with practically the same symptoms as on admission.

Discharge Diagnosis.—Brain tumor was suspected; hysteria.

Comment.—It was considered most probable that there was a circumscribed arachnoiditis in the angle. Acoustic tumor was possible since such cases often give a history of chronic otitis media. At least

one good result came of the operation. She was cured of having operations.

Some time after operation a friend came to visit the patient. For apparently no reason the patient called me aside and told me not to talk to the woman for she was a great liar. Of course the friend was interviewed as soon as possible. She stated that she and the patient had lived alone for some years, and although she did not consider the patient insane, she thought the patient was never normal. Many times she tied the patient in bed at night to keep her from walking away. Attacks of semiconsciousness and speechlessness had been present several years previous to the first mastoid operation. The friend could always tell when an attack was coming on by the listless way the patient sat around. The relatives denied all knowledge of such occurrences.⁸

C. Clinically, Tumor.—In a single instance the diagnosis was not doubted, clinically, but no evidence of tumor was disclosed although the region was fully exposed. It seems equally certain now that no tumor was present, but necropsy examination is necessary for verification. The case follows:

CASE 8 (P.B.B.H. Surg. No. 10727).—*Dyspituitarism with diabetes insipidus. Operation. Negative findings.*

June 27, 1919, David S., aged 14½, a schoolboy, recommended by Dr. Jacob Rosenbloom of Pittsburgh, was admitted to the hospital with complaint of poor vision and excessive thirst.

Chronology of Symptoms.—For the last ten or eleven years the patient had suffered from excessive thirst and polyuria. Sight had been failing for two years.

Positive Findings.—The findings were: bilateral primary optic atrophy; visual acuity about 10/50 in each eye with a very small area of vision in the lower part of each field. The clinoid processes were greatly thickened. There were slight skeletal undergrowth and feminine distribution of hair, but the genitalia were well developed and the voice was quite masculine. Libido was undeveloped; basal metabolism, —16; average water intake per diem, 8,500 c.c.; daily urine output about the same. The body temperature was very variable, 95 to 99 F.

Clinical Diagnosis.—A diagnosis of supracellar cyst was made.

Operation.—July 14, 1919, the usual transfrontal procedure was carried out with no difficulty. The frontal lobe was easily elevated, and a good view was had of the chiasm. No evidence of any tumor was disclosed.

Postoperative Notes.—The condition remained unchanged until he was discharged July 31, 1919.

Discharge Diagnosis.—Brain tumor was suspected; diabetes insipidus.⁹

8. Note from the patient Oct. 24, 1920, states: "Dear Sir: In reply to your request of October 4, will say . . . that I am able to work and as far as your records are concerned—condition is O.K."

9. A note from patient's sister, Oct. 8, 1920, stated: "We can notice no material improvement in his condition. His health is about the same, that is, it is very good in comparison to the condition of his eyes. He still maintains an enormous consumption of water"

Comment.—In a very similar case, except for the diabetes, a cholesteatoma of the third ventricle was found.¹⁰

D. Verified Nontumors.—In 1904, Nonne¹¹ described a series of cases which he tried to combine into a clinical entity characterized by more or less slow development of the picture of brain tumor. If death occurred, neither macroscopically nor microscopically was any adequate lesion discovered. To such a clinical picture, Nonne applied the term "pseudotumor cerebri."

Many similar cases have subsequently been reported, but always, when properly examined, definite histopathologic changes have been found and these of the most varied description. As has been pointed out elsewhere,³ the use of the term "pseudotumor" to refer to them is unfortunate. Malaria and miliary tuberculosis may simulate typhoid fever but to designate such instances as "pseudotyphoid" would only cause confusion. It is certain from a careful examination of lesions simulating tumor of the brain that no such clinical nor pathologic entity exists as Nonne tried to establish. Furthermore, tumor of the cerebellum is simulated at least as often as tumor of the cerebrum.

It has been decided in this clinic to discard the term "pseudotumor cerebri" although it is occasionally written in parenthesis after one of the brain tumor suspects to indicate that it might be such a condition as Nonne had in mind.

The rules for recognizing pseudotumors laid down by Nonne,¹² in his last monograph on the subject, are copied from Higier as follows:

1. Evident infection, severe anemia, and physical and psychic trauma must be absent.
2. The onset must not be acute.
3. Signs of essential hydrocephalus must be absent (head deformity, etc.).
4. If recovery occurs, it must be gradual.
5. If death occurs, no pathologic changes must be found after thorough microscopic study.

There may have been cases at the time of Nonne's first publication (1904) in which no pathologic changes could be demonstrated, because the methods used were imperfect. Certainly every case examined by modern histopathologic methods, especially those for bringing out changes in the glia, has shown definite lesions.

10. Bailey, P.: Cruveilhier's "Tumeurs Perlées," *Surg. Gynec. & Obstet.* **31**:390, 1920.

11. Nonne: Ueber Fälle vom Symptomencomplex von Tumor cerebri mit Ausgang in Heilung, *Deutsche Zeitschr. f. Nervenheilk.*, **27**:169, 1904.

12. Nonne: Der Pseudotumor cerebri, *Neue deutsch. Chir.* **10**:107, 1914. (Die allgemeine Chirurgie der Gehirnkrankheiten, Part 2.)

In the present series were two patients who had typical history and findings of localizable tumor. Concerning one, Lizabel McN., Surg. No. 11187, recorded in detail elsewhere,³ there will be no question. There was present a complete picture of tumor of the left cerebellar hemisphere. Histopathologic examination of the brain proved it to be a meningoencephalitis.

The history of the other case will be given and the propriety of its inclusion in this group discussed later.

CASE 9 (P.B.B.H. Surg. No. 10912).—Solitary tubercle of left cerebellar hemisphere. Removed at operation. Tuberculous meningitis. Death.

July 30, 1919, Stewart D., aged 27, a business man, was admitted to the hospital, recommended by Dr. Hoover of Cleveland, Ohio. He complained of headaches, vomiting and unsteady gait. The left testis and epididymis were removed in 1916 for tuberculosis.

Chronology of Symptoms.—For six months he has suffered with stiffness of the neck, for four months with dizziness and vomiting on arising; for three months with suboccipital pain, and for two weeks with a tendency to deviate to the right in walking and with unsteadiness of the right hand.

Positive Findings.—There were present: bilateral choked disk of I D.; marked nystagmus on looking to right and left and a positive Romberg sign. The gait was staggering. Slight ataxia was found in both hands, more noticeable in the left. There were also slight left facial weakness, suboccipital tenderness on both sides; the head was held tilted to right. Bradycardia was found.

Operation.—Aug. 1, 1919, under procain anesthesia a large solitary tubercle of left cerebellar hemisphere adherent to the dura was disclosed. It was removed intact by careful dissection with ample margin of brain tissue.

Postoperative Notes.—On August 17, the temperature was normal and the patient felt perfectly well. On August 23, there was afternoon temperature of 100 F., and some stiffness and soreness of neck. On September 11, the temperature was falling; on Sept. 14, the patient became irrational.

Second Operation.—A parasagittal incision was made, September 16, into the old operative defect in bone on left side revealing a tuberculous meningitis. On September 19, the temperature was normal, the patient being irrational and incontinent. On September 22, the temperature was 97 F. The patient died September 24 with temperature 105.4 F.

Necropsy.—This was performed September 25, and revealed tuberculous meningitis, a small tuberculous cavity in apex of right lung and tuberculosis of prostate.

Discharge Diagnosis.—The diagnosis was tuberculosis of the prostate and right lung, tuberculoma of left cerebellar hemisphere, postoperative tuberculous meningitis.

Comment.—The possibility of tubercle was not overlooked here, although physical and roentgen-ray examinations of the chest were entirely negative. At any rate, surgical relief was indicated, for whether tubercle or tumor it was evidently interfering seriously with cerebrospinal fluid drainage.

There is some question as to whether tuberculoma and gumma should be included under the brain tumors. There is no doubt that

both may give rise to typical tumor manifestations and even demand surgical removal. On the other hand, neither tubercle nor gumma is a neoplasm, but rather an infectious granuloma and the indications for surgical therapy are far from clear in many cases.

Although a solitary tubercle may be successfully removed, there is no way of knowing that others do not exist in the vicinity. If the surgeon cuts into one of these, infection of the meninges is almost certain. Even in the most favorable cases, such as the one just reported in which necropsy proved that only a single large tubercle was present, the result is very often utterly discouraging. Gummas may be much more safely removed, but it is rarely necessary. Certain old, hard, fibrous lesions may necessitate surgical removal, but most of them may be successfully combated by medical means.

These are border line conditions, and it is very difficult at times to know just where to draw the line. Multiple small tubercles may be present. Also, the astonishing thickening of the basilar meninges encountered at times in syphilitic basilar meningitis may simulate very closely suprasellar tumor. In order to emphasize the problems connected with these lesions, there is a tendency in this clinic to group the infectious granulomas with the tumor suspects.

II. UNVERIFIED BRAIN TUMORS: TWENTY-FOUR CASES, 20.34 PER CENT.

The unverified brain tumors fall definitely into three groups. The meaning of the term "verification" will be explained under Group III.

A. Unoperated.—Of the thirteen unoperated cases, five operations were postponed merely for the convenience of the patient; four cases were thought to be pontine gliomas for which no operative procedure is of any avail; two were metastatic carcinomas; one patient was an acromegalic whose symptoms had been quiescent for twenty years; and in one case, probably a frontal glioma, the patient refused operation. In every case both pressure and localizing symptoms were present (except the pituitary cases in which pressure symptoms were absent).

B. Unlocalized.—Five patients had definite pressure syndromes but insufficient localizing symptoms. In such cases, a right subtemporal decompression was usually performed to relieve pressure and particularly to save vision menaced by choked disk, a procedure now well established in cranial surgery. In two of these cases, a large bone flap was turned down and converted into a decompression when no lesion was disclosed. At any rate, a definite attempt to reach a tumor was not made.

C. Undisclosed.—Six times a definite attempt was made to remove a tumor, three times by a suboccipital approach, twice by a boneflap and once by a transsphenoidal approach.¹³ The lesions were not disclosed, and although in two cases in which a boneflap was turned down a cerebral glioma was certainly present, since a fragment was not removed for histologic study, these cases could not be listed as verified. Also by a suboccipital approach, it was impossible to verify a deep lying cerebellar glioma which had not broken down and become cystic. That the difference between verification and nonverification may depend on a very small thing is shown in the case of P. P. (Surg. No. 10883) in which a fortunate puncture deep into the cerebellum struck a small gliomatous cyst from which a few drops of characteristic yellow fluid were secured. He would otherwise have been included in this group. On the other hand there has been already noted under Group I, *D* the case of L. McN. (Surg. No. 11187) originally placed in this group, who came to necropsy after this article was begun.

The cases in Group II are not so interesting as those in Group I. The presence of tumor was not doubted from clinical examinations, and during the operation nothing occurred to cast suspicion on the diagnosis.

III. VERIFIED BRAIN TUMORS: SIXTY-THREE CASES, 53.39
PER CENT.

This group will be dismissed with a few remarks. A number of cases have been already noted. They present few difficulties in classification except for the pathologist, and with the difficulties of pathologic diagnosis we are not here concerned. It will be noted that although the tumors verified include only 53.39 per cent. of all the cases quoted in this series, if we include only Groups II and III, they constitute 72.4 per cent. of the actual tumor cases (so far as it is possible to estimate the exact number at the present time). If we again exclude the unoperated cases in Group II, *A*, 85.13 per cent. of the operated tumor cases were verified. The members of this group were distributed pathologically as in the accompanying tabulation.

It is, of course, apparent that, in nearly two thirds of the cases, symptoms referable to the brain arose solely from the fact that the structures from which the tumors originated were enclosed in the cranium along with the brain. It will be observed that in this series the gliomas alone arose from the brain tissue. As has been often

13. This patient suddenly became unconscious, Sept. 15, 1920, while at work. He was taken to the Boston City Hospital where he died in a few hours. At necropsy there was found a tumor of the pituitary gland which had dissected its way extensively beneath the dura into the temporal fossae.

stated before it would be well to abandon the term "brain tumor" for the more accurate and suggestive term "intracranial tumor."

A. Verified at Operation.—Tumors were never classified as verified at operation without removal of a fragment for histologic examination, with one exception—the straw-colored fluid which coagulates on standing, contained in a gliomatous cyst, can scarcely be mistaken for anything else.

PATHOLOGIC DISTRIBUTION OF CASES OF VERIFIED BRAIN TUMOR

	Cerebral						Pituitary		Cerebellar		Brain Stem		Total
	Frontal	Precentral	Occipital	Temporo-sphenoidal	Basal	Cavum Meckel	Intrasellar	Suprasellar	Intracerebellar	Extracerebellar	Pontine	Medullary	
Gliomas.....	1	3	1	5	1	12	1	24
Endotheliomas..	2	3	2	2	..	1	..	2	12
Neuromas.....	6	6
(acoustic)													
Adamantinoma.	1	1
Carcinoma.....	1	1
Tumeurs perlées	1	..	1	2
Adenomas.....	11	11
(pituitary)													
Congenital cysts	2	2	4
Epithelioma.....	1	1
(Rathke's pouch)													
Papilloma.....	1	1
(choroid plexus)													
Total.....	3	6	3	7	2	3	13	6	12	7	0	1	63

Many of the cases, except for accident, would of necessity have been classified as unverified. One case in point is E. T. (Surg. No. 10572). A glioma of the medulla oblongata was exposed at operation. It was evidently impossible to remove a piece of tumor for histologic examination although the nature of the condition was unquestionable. The patient subsequently died of respiratory paralysis and the history came up for discussion before the pathologist's report was received. I proposed that the case be put in Group III *A* as a tumor verified at operation, but Dr. Cushing protested. When the diagnosis of glioma was established by histologic examination of the necropsy specimen, and then only, was the case classified in Group III *D*.

B. Verified at Necropsy.—The single unoperated case is noteworthy for the reason that had necropsy been refused it would have been relegated to the group of suspects.

CASE 10 (P.B.B.H. Med. No. 11985).—*Abscess of the frontal lobe, with concurrent endothelioma. Death. Necropsy.*

Oct. 8, 1919, Frederick S., aged 50, an optician, recommended by Dr. Belle, of Waltham, Mass., was admitted complaining of "brain tumor."

Chronology of Symptoms.—For one year, occasional trancelike states had occurred at night. The patient would wake up and feel he could not move. For thirteen weeks there had been pain in the upper teeth on the left side spreading along to the eye and then to the head, lately it had been very severe in the left temporal region and constant, keeping him awake at night. Four weeks previously the left upper molar tooth had been removed for abscess at the root. For one week there had been occasional vomiting with slight nausea, photophobia, flashes before the eyes and failing vision. For five days he had suffered difficulty in swallowing and speaking and disturbance of the sense of smell and taste. For four days there had been rigidity of the neck and for two days he had been totally blind.

Positive Findings.—The rectal temperature was 99.8 F. There was rigidity of the neck. The reflexes were hyperactive. The pupils dilated without reaction to light. There were right external rectus paresis; complete left ophthalmoplegia and profound stupor. The right optic disk was edematous, hyperemic and elevated $\frac{1}{2}$ D, the left was elevated 1 D. The leukocyte count was 18,000.

Course.—October 9, poorly sustained ankle clonus was observed on the right side and positive Kernig sign on the left. October 12, transillumination of the sinuses was negative. October 16, the findings were ptosis of the left eyelid, ankle clonus on both sides and normal temperature. October 18, lumbar puncture revealed straw-colored fluid with 465 polymorphonuclear leukocytes per cubic mm. October 19, the patient died.

Necropsy.—This revealed dural endothelioma in the region of the sphenoidal wing in the left middle fossa; abscess of the left frontal lobe; purulent meningitis and suppuration of the left maxillary and sphenoidal sinuses.

Comment.—The whole course of this case distracted one's attention from the possibility of tumor. The only clue we get in the history is furnished by the story of peculiar trancelike states for a year previous to admission. Such states accompanied by a sense of unreality are well known in temporal lobe tumors and in this case were doubtless due to the tumor.

C. Verified at Operation and Necropsy.—The single case verified at operation, which subsequently came to necropsy (C. M., Surg. No. 11078), was an extensive glioma of the left cerebral hemisphere. This patient had been previously operated on by Sir Victor Horsley, presumably for jacksonian epilepsy.

D. Verified at Necropsy, Unverified at Operation.—Of the four cases unverified at operation and verified later at necropsy, one (M. C., Surg. No. 10252) was an extensive glioma of the thalamus with an advanced secondary hydrocephalus; another patient (T. H., Surg. No. 11462), had a bilateral papilloma of the lateral choroid plexuses; a third (E. T., Surg. No. 10572) an extensive glioma of the medulla oblongata; and the fourth (A. B., Surg. No. 10231) an extensive glioma of the right frontal lobe whose only symptoms were those of a suprasellar tumor. A transfrontal procedure was adopted with the last patient. The findings were negative, and death occurred some weeks subsequently at her home, the brain being sent to us by her physician.

Finally, it is important to call attention to the fact that in the last group of tumors, verified, the clinical picture was often no clearer than in the cases given under Group I, *B* or Group II, *C*. The following is an unusually valuable case in point.

CASE 11 (P.B.B.H. Surg. No. 10681).—*Endothelioma of left cerebral hemisphere with flaccid paralysis of right arm. Extirpation. Recovery.*

June 20, 1919, Dorothy R., aged 22, a student, was admitted complaining of paralysis of the right arm, weakness of the right leg and headache.

There had been panophthalmitis in the right eye, at 10 years of age, following an injury, accompanied by sympathetic inflammation of the other eye. Vision was much obscured in each eye.

Chronology of Symptoms.—In 1914, there were attacks of peculiar sensation, like an electric shock in the right hand. Later attacks involved the right arm and face. September, 1918, motor weakness began in the right arm. For one month, there was weakness of the right foot; for one month, headaches; for one week, vomiting following headaches. There was a vague history of rare attacks of twitching in the right arm elicited after much questioning.

Positive Findings.—No sign of sensory defect was detected except absence of abdominal reflexes on the right side. There was marked hypotonicity of the musculature of the entire right half of the body, most marked in the right arm, which showed total flaccid paralysis except for slight movement in the biceps and deltoid. Motor weakness, so marked in the arm, was slight in the leg and barely perceptible in the face. Tendon reflexes were exaggerated on the right side, very slightly exaggerated in the arm, more so in the leg. There was a suggestive Babinski sign and a poorly sustained ankle clonus on the right. There was atrophy of the interossei muscles of the right hand. Accurate ophthalmoscopic and perimetric observations were impossible because of cloudiness of the media from old inflammation.

Clinical Diagnosis.—Endothelioma of the left precentral region was the diagnosis.

Operation.—Osteoplastic exposure of the left cerebral hemisphere was performed July 8, 1919. A 32 gm. nodular endothelioma was removed from the precentral area.

Discharge Diagnosis.—Brain tumor was verified: endothelioma.

Comment.—This patient had been treated by an excellent neurologist for a "neurosis" and by another for an "occupational neuritis." She was at that time studying with a famous pianist. The total absence of spasticity, the marked hypotonicity, the absence of sensory signs, the vagueness of the history of motor attacks elicited after questioning and possibly due to suggestion, the slight but unmistakable atrophy of the interossei of the right hand, the absence of subjective symptoms of headache and vomiting since admission, the difficulty of making proper ophthalmoscopic examination, all these things made it very hard to come to a decision in this case. Furthermore, she was the only child of very evidently psychopathic parents. She was kept under observation for some time, and operation was finally decided on as a result

of exacerbation of headaches and vomiting, coupled with the presence of a Babinski sign and ankle clonus in the right foot. Undoubtedly, if she had come to us a month earlier, before headache and vomiting had appeared and possibly before the development of motor signs in the right foot, no operation would have been undertaken.

SUMMARY

There has been given an account of the method used to classify clinically patients having, or supposed to have, intracranial tumors, in the hope that it will be of value in interpreting statistics from this clinic and perhaps assist in unifying general usage in this regard.

My thanks are due to Dr. Harvey Cushing for permission to publish these cases as well as for valuable suggestions in the preparation of this article.

THE DANDY METHOD OF LOCALIZING BRAIN TUMORS BY THE ROENTGEN RAY

WITH REPORT OF A CASE

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TOPEKA, KAN.

The indispensability of roentgen-ray examinations in general medicine is well recognized. Few physicians realize, however, how much assistance the roentgen-ray picture of the skull and of the spine can afford the neurologist and the psychiatrist. Certain cases of congenital brain syphilis, of traumatic epilepsy, of pituitary disease, of cerebral arteriosclerosis, of cervical rib and of a few other conditions have, in our experience, been diagnosticated chiefly on the basis of the roentgen-ray findings.

Nothing is more disappointing, on the other hand, than the roentgen-ray findings in brain tumor. There are few cases of brain tumor in which the roentgen ray gives us any information other than that which we already possess from clinical findings. It is usually of no help in localization.

All of this is, perhaps, on the point of being radically changed by the principle of photographing the ventricles of the brain filled with air. This idea was suggested and put into practice by Walter E. Dandy of Baltimore, who showed that when ventricular fluid was removed and air injected, a roentgen-ray plate showed sharply the outlines of the ventricles. A tumor present in the brain is likely to encroach on the ventricle at some point or another and this distortion would clearly show in such a roentgen-ray photograph. The procedure, then, is to make a small trephine opening in the skull, insert a long needle into the ventricle, remove the cerebral fluid, inject air, photograph with the roentgen ray, and compare with similar photographs of normal ventricles.

The present opportunity is much too limited to present the entire theory of the interpretation of these plates. This has been done by Dandy in six or seven contributions, and the procedure will be exemplified by a case of my own.

I am quite aware that the following case is not a perfect illustration of the advantages of Dandy's method. While there is apparent correlation of the clinical diagnosis and roentgen-ray findings, it is a point of great regret that surgery did not confirm the diagnosis and effect the treatment. I think we should not be the least disheartened

by this for the reason that in all human probability there will long remain problems of treatment even after the diagnosis is indisputably made. The attempt at surgical removal in this case was made, and the fact that it was unsuccessful is not a reflection on the surgery, for such failure has long been a common surgical experience; nor, on the other



Fig. 1.—Stereoscopic illustration showing no abnormality except a shallow sella and evidences of tremendous intracranial pressure in the separation of the sutures.

hand, is it a reflection on the diagnosis, for there is little doubt, everything considered, about the correctness of this. On the whole, there is reason to believe that this method will be of tremendous value in the more accurate localization of brain tumors.

CASE REPORT

History.—F. S., Case 206, was a lad of 6 referred by Dr. J. F. Shelley of Elmdale, Kan. His family history was negative except for the following facts: A second cousin of the father was said to have been insane and to have had epilepsy. The father also had a sister subject to sick headaches. The father was of English descent, the mother of mixed ancestry. Both were fairly typical



Fig. 2.—Left side of the head. The ventricle has been filled with air. It is not encroached on at any point but is uniformly dilated to about three times the normal size.

rural folk, aged 44 and 42, respectively. There were three other children, aged 11, 4 years, and 6 months, all in good health.

The child was born at full term, normal delivery, was breast fed and began walking, talking and teething at the usual ages. He had the nail-biting habit, but there was no history of convulsions, enuresis, etc. He had always been

well, except for occasional attacks, when he would wake up with a headache, vomit and then feel all right. He had had several accidents, such as falling off of a hay wagon and being run over by a lumber wagon, but with no ill effects so far as known. Although he had not been to school, he had learned to count to 20 with a few mistakes. His social and temperamental reactions were normal.

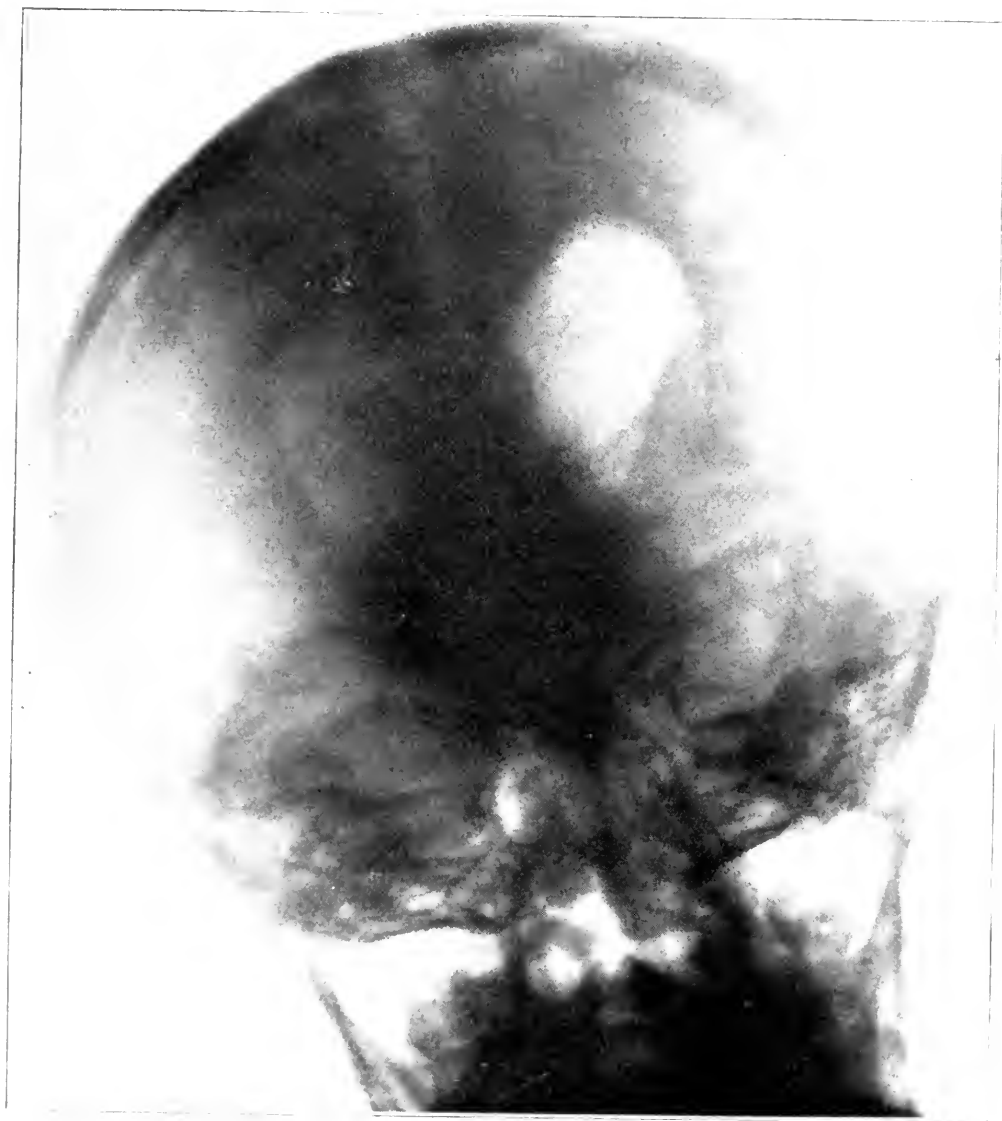


Fig. 3.—An anterior-posterior view of the head after the injection of air. This picture was taken with the subject in a prone position.

Present Illness.—In November, 1919, he had had scarlet fever of moderate severity, followed by slight albuminuria. Headaches of marked severity, especially over the frontal regions, were noticed shortly afterward. Headaches had

been noticed for several years as mentioned in the foregoing, but they now became prominent and occurred every day, and were more severe at some times than at others. Vomiting nearly always occurred in association with them. It was not projectile and did not always relieve the headache. The urine was negative. He was given some salicylates and seemed to improve.

Nearly three months later he was again brought to the physician's office with the report that the headaches had increased in severity. For two weeks he had been holding his head tipped to the left, and he once complained of not seeing well. Although he had not fainted or fallen, he had difficulty in walking and the physician thought there was a tendency to veer to the left. He was untidy at the table and was restless and fidgety all the time. He had had no fever.

Physical Examination.—He was first seen by me on April 19, 1920. At that time I made the following notes: bilateral choked disks; pupillary reaction normal but left pupil irregular in outline; visual acuity not greatly diminished; head tipped to the left; slight left facial paresis; tongue and uvula deflected to the left; positive Macewen's sign; no speech defect; hypotonus of the fingers, especially of the left hand; reflexes generally normal; positive Romberg sign; bilateral Babinski and Schäfer's reflex; erection of penis when abdomen is scratched, and staggering gait. Special cerebellar tests were made following the schemes of Weisenburg, Tilney, and others.

The Bárány tests of past-pointing were used. With the right hand there was almost always a spontaneous deviation to the left of from 5 to 20 cm., usually about 10. This was true whether the horizontal or the vertical plane was used. With the left hand the deviation was not quite so constant nor quite so marked, but there was some deviation to the left apparent with both planes. Stimulation of the internal ear was not done.

Babinski's five tests of asynergia minor were nearly all positive. The leg was raised in two movements instead of one, the body was flexed before the leg in attempts to sit up (in fact, the legs were never flexed, but were raised stiffly with the buttocks as a pivot). Touching points with his toes was inadequately and jerkily done. Kneeling on a chair was not tried. He was unable to lean backward and failed to make proper coordination of the legs when it was attempted.

Andre-Thomas tests for dysmetria were made. The grasping of a glass of water, the supination of the palms and the awkward heel to knee placing were suggestive but not convincing. Babinski's adiokokineses tests were distinctive, the left arm being much worse than the right. The rebound phenomenon of Holmes was slightly present on both arms.

Trunkal asynergia was manifested in the saddle gait which showed cerebral walking rather than cerebellar. He could sit for a long time and had more difficulty with his eyes shut than with them open. In other words, he seemed to have a positive Romberg sign. Fournier's tests of gait were found inconclusive.

Laboratory Findings.—The blood and urine were normal. The spinal fluid pressure was greatly increased. The globulin test was positive; the Wassermann and colloidal gold tests were negative.

Roentgen-Ray Examination.—Stereoscopic roentgen-ray examination (before trephining) of both the right and left sides of the head showed marked evidence of intracranial pressure. The suture lines were wide open, especially in the region of the anterior fontanel. There was no evidence of a sella.

Visual Fields.—The visual fields were studied by Dr. C. L. Williams on April 9 and showed marked contraction, approximately equal on both sides, and in both instances averaging only about 15 degrees. They were fairly round.

Treatment and Course.—On April 22 the spinal fluid was secured. It was under great pressure and following the removal of a small amount the choked disk receded noticeably and the facial and eleventh nerve palsies largely disappeared. His gait was better than before.

On May 11 a small trephine opening was made at the vertex, the ventricle tapped by means of a lumbar puncture needle and 50 c.c. of cerebral fluid removed, the same amount of air being injected gradually as small quantities (10 c.c. at a time) of the fluid were removed. Roentgen-ray plates were made before the patient came out from under the anesthetic. There was a slight reaction of fever and vomiting, but this was not serious and disappeared after thirty-six hours. The patient was much better after this. Splashing of the cerebral fluid could be heard at a distance of several feet when his head was shaken.

Nystagmus was detected for the first time on May 14, although it had previously been looked for. When the eyes were directed to the right there was a definite nystagmus with a slow component to the left; when the eyes were directed to the left there was a nystagmus with a slow component directed to the right.

As the roentgenograms show, the diagnosis of cerebellar tumor was adequately justified. In the first place, there was an obvious hydrocephalus. This is shown by the lateral views. The anterior-posterior view taken with the child in the prone position, shows that the two lateral ventricles were dilated symmetrically and equally. Fluid removed from the left ventricle must drain through the third ventricle into the right ventricle, into which the needle was inserted, and this illustration shows that the drainage was apparently incomplete. That it was a technical fault appears probable from the fact that the ventricle did receive enough air to outline it distinctly, and the haziness is caused by the pool of fluid in the anterior horn of the ventricle. The approximate equality in dilatation of the two ventricles and the fact that they are not displaced from the midline shows, according to Dandy, that the obstruction (neoplasm) is cerebellar, between the aqueduct (or foramen of Monro) and the foramen of Magendie.¹

I am indebted to Dr. Samuel T. Orton for the suggestion that this statement of anatomico-pathologic principle is scarcely broad enough. Any obliterative focal pressure exerted on the aqueduct of Sylvius behind the anterior end would cause the same result (equilateral hydrocephalus). That this need not be restricted to cerebellar tumors must be obvious from a consideration of the outlines of this region. Tumors springing from the corpora quadrigemina, for example, or from almost any point in the floor of the fourth ventricle, could and likely would

1. Dandy says: "Except in rare instances only tumors in the brain stem or cerebellum can produce a symmetrical bilateral internal hydrocephalus." Surg., Gynec. & Obst., April, 1920, p. 338.

produce the same picture, and of course, an inflammatory process with blocking of the aqueduct would produce the same effect, as Dandy² himself has illustrated.

We need, then, to look well to the clinical (neurologic) indexes, and if these fail, it is doubtful that we are justified in positively concluding that the lesion is a cerebellar one.

Further Course.—In this particular case the neurologic indications seemed well confirmed by the ventriculographic plates (or vice versa). Operation was attempted a few days later. A bone flap was reflected in the occipital region, and neither inspection nor palpation revealed a tumor. The patient reacted poorly to the anesthetic. Constant blood pressure readings were made, and after about thirty minutes, the rising pulse and falling pressure made it seem wise to discontinue the search, and the wound was closed.

The patient made a poor recovery from the operation and was for many days stupid and at times delirious. He had a few convulsions during the second week. He gradually regained strength and was removed from the hospital. He has not been seen since.

2. Dandy: The Diagnosis and Treatment of Hydrocephalus Resulting from Stricture of the Aqueduct of Sylvius, *Surg., Gynec. & Obst.*, October, 1920.

News and Comment

DR. PEARCE BAILEY RECEIVES DISTINGUISHED SERVICE MEDAL

Dr. Pearce Bailey, New York, who was chief of the Division of Neuro-psychiatry in the Surgeon-General's Office during the World War, has been awarded the distinguished service medal. The citation is now in the hands of Surgeon-General Ireland and will shortly be presented to Dr. Bailey at his home.

AMERICAN NEUROLOGICAL ASSOCIATION MEETING

The next meeting of the American Neurological Association will be held in Atlantic City, N. J., in the Hotel Chelsea, on June 13, 14 and 15, 1921.

Abstracts from Current Literature

SUR UNE FORME BENIGNE DU SYNDROME DE BROWN-SEQUARD
(A BENIGN FORM OF THE SYNDROME OF BROWN-SEQUARD).
S. GOLDFLAM, *Rev. neurol.* **35**:673 (Sept.) 1919.

The author reviews briefly the history of the diagnosis and treatment of Brown-Séquard cases: he says that, at first every case not definitely due to trauma or hematomyelia was regarded as syphilitic, and that one must be constantly on guard against forgetting this etiologic factor. He then speaks of how, in the past twenty years, especially, cord tumors have come to be recognized as etiologic of the syndrome. Then there came a time when it was regarded as a grave error if exploratory laminectomies were not made in every suspected case of cord tumor, this being followed by the inevitable reaction to this attitude because of serious postlaminectomeal results. Then when, instead of a tumor, one found at operation increase of fluid between the membranes resulting in pressure symptoms, there arose such classifications as meningitis spinalis, serosa chronica circumscripta, subarachnitis chronicans circumscripta, or so-called "meningeal cyst." If such a cyst appeared above or below a cord tumor, exact localization became all the more difficult. Even at the present time it is difficult and, at times, impossible, to differentiate between a meningeal cyst and a tumor. Frequently the symptoms are as varied and atypical as in multiple sclerosis. This is especially true of multiple sclerosis at the sacral level of the cord when the Brown-Séquard syndrome is present. Then there are (as demonstrated by Oppenheim) cases which present symptoms of cord tumor where the primary lesion is of an inflammatory nature involving the conus medullaris and the cauda equina.

It is not a rare thing to have all the clinical symptoms of a tumor of the cauda equina, and yet on operation fail to find anything abnormal, either microscopically or macroscopically. There are cases such as those reported by Nonne and Freud in which necropsy revealed inflammatory conditions of funicular myelitis instead of tumors. Nonne and Freud also repudiated the idea popularized by Lichtheim that funicular myelitis was fatal; instead, according to them, the condition may remain stationary or even regress. Fleischman is of the opinion that there are benign and even abortive forms of myelitis. This he thinks is especially true in cases of alcoholic etiology.

The former opinion that the Brown-Séquard syndrome was indicative of a progressive and fatal course has given way to a more hopeful attitude in the light of the recovery of many patients with typical cases. Boettiger reports a case in which the patient was entirely cured at the end of ten years.

Oppenheim was the first to call attention to a form of the Brown-Séquard syndrome which occurred chiefly in men between the ages of 40 and 50.

The clinical picture is usually the same, with the typical Brown-Séquard syndrome: dorsal localization, bladder trouble and usually impotence. The patient can usually walk. There is hypertonia, exaggerated reflexes and the Babinski sign. There is also pronounced weakness of the opposite leg. There is usually the reflex of Rossolimo or some spastic phenomenon. Sensory disturbances are variable, pain being present in great degree or totally absent, by preference on the dorsal aspect of the foot. Girdle pains are exceptional;

radicular pains never present. The status of paresthesia and anesthesia is almost as variable. Sensory disturbances usually appear on both sides of the ventral aspect of the trunk. Oppenheim regards the evolution of the syndrome as most important, and the following is typical: the lesion begins in the subacute form and reaches the crisis within a few months, although in some cases it may last for years. The process may be ameliorated or arrested, but never completely cured.

The author then reverts to an article of his own which appeared as early as 1908, in which he used the phrase, "a benign form of the Brown-Séquard syndrome." He then adds that some of these cases not only show a tendency to amelioration, but even complete cure. He bases this opinion on cases that have run from four to ten years, and reports five cases.

The five cases reported are interesting in that the patients all were robust men between the ages of 29 and 50, none had syphilis or tuberculosis, and none showed an infectious-toxic process, such as that of alcohol or pernicious anemia. The clinical picture for the most part is uniform, that of the usual Brown-Séquard syndrome. One of the most striking symptoms was the loss of sexual power. Vesical symptoms were uniformly mild. The author then goes into detail relative to the various symptoms, one of his most significant remarks being that the phase of sensory disturbance is synchronous with that of disturbance of motility. His résumé of his cases includes a discussion of what he calls the period of progression, the stationary period, and the period of regression. The period of regression is often interrupted by relapses which may last for months or for years. Bladder and bowel symptoms may disappear entirely. Paresthesias may become less marked, but impotency usually persists. Amelioration seems to persist. One may speak of cures in the sense that the patient attends to or returns to his occupation learning to ignore his milder symptoms. But there are true relapses, with, perhaps, exacerbation of all the symptoms, especially in the field of motility.

To sum up: It may be said that the characteristic element of the Brown-Séquard syndrome is the slow insidious development of the symptoms without apparent etiology in men of middle age. Even at the height of the illness the symptoms are not severe, but the clinical examination shows that they are not functional, but rather weakness or hemiparesis of the spinal type. There are never symptoms in the upper extremities or of the cranial nerves. The second or stationary phase may last for years, after which there may supervene a phase of regression of equally long duration. In all the phases there may be recrudescences. Cure is durable though there may persist insignificant symptoms.

The author believes that the benign form of this syndrome is more frequent than cord tumors. It is necessary to give more attention to the differential diagnosis and keep in mind the following symptoms: slow onset, slow development, chronicity, preponderance of paresthesias (especially thermic in one leg), absence of intense pain, etc. On the other hand, one must not forget the difference between the insignificant subjective symptoms and those revealed by objective examination which establishes the lesion in the dorsal portion of the cord. It is also necessary to exclude syphilis, especially gumma localized in the dorsal portion of the cord. In the latter, the course is not so steady, and is amenable to antisyphilitic treatment whereas in the benign form of the Brown-Séquard syndrome, such treatment has no favorable result. Syphilis of the cord, not treated, is almost always fatal; sooner or later complete paralysis, retention of urine, cystitis and all of its consequences follow.

Multiple sclerosis must also be excluded. Here the diffuseness of the lesions is characteristic. The most difficult task is to rule out spinal cord tumors. Because of the great prognostic difference, the author discusses the question as to whether laminectomy is justifiable. Even when a tumor has not been found, it has been said improvement frequently follows decompression, and even when we cannot say just why. The writer cites one case of a patient, who died three weeks after operative procedure, as an example of the necessity of making a careful differential diagnosis. Tumors of the meninges and intramedullary tumors cause the most difficulty.

It is during the first phase of such cases as those of the author's that the greatest diagnostic difficulties arise, when it is difficult to rule out tumor of the cord, not the typical meningeal tumor with characteristic root pains, but those in which the symptoms are comparatively mild.

The difficulties of diagnosis increase when we have atypical cases of meningeal tumor, when there is little pain, or when the root pains are absent and displaced by paresthesias. Cord tumors rarely show medullary symptoms (sensory and motor), except at a rather late stage, whereas in the type of case discussed by the author, such appear early.

In cord tumors, the motor symptoms usually appear earlier, whereas in these cases of the benign form of the Brown-Séquard syndrome, the motor and sensory symptoms may be of equal intensity, although the sensory symptoms are usually the more pronounced. Weakness appears early, whereas in cord tumors, it is late. In cord tumors the symptoms of hemiparesis rarely form an episode; they are usually abrupt in their onset and usually are progressive to the terminal stage.

Remissions are rare, whereas in the benign form under discussion, remission is usually one of the most pronounced features in the clinical picture. The remission may be followed by a relapse, but this in turn by another remission. In cases of cord tumors, this is not true, and the relapse is often dangerous to the life of the patient, and the periodicity of the relapse is part of the clinical picture in tumor cases. In the latter the development is usually not as gradual as in the author's so-called "benign" cases. It is the author's opinion that too much emphasis must not be laid on the "syndrome compression" (increase of globulin, frequent xanthochromia, and lack of pleocytosis in the spinal fluid) as diagnostic of cord tumors. He cites Oppenheim's experience in this respect.

On the other hand, the writer lays the greatest emphasis on the necessity of making a sharp differential diagnosis to the end that one may know when surgical procedure is imperative. He lays down the axiom, that in tumor cases, the patient must not be allowed to pass what he styles the "second phase"; that is, the phase of the Brown-Séquard syndrome. Laminectomy should always be resorted to before this stage is past.

When in atypical cases we notice a steadily progressive course with accentuation of motor symptoms, we know that we are approaching the period or phase of transverse lesion. This is the danger signal and surgery is imperative. These observations on differential points in diagnosis hold just as truly for so-called arachnitis serosa circumscripta spinalis as for cord tumors. He believes that there is little difference between the symptomatology of the cystic tumor and that of the solid tumor.

He then discusses Boettiger's adoption of the term "pseudomedullary tumor" as analogous with "pseudocerebral tumor," and as applied to those supposed tumors in which there followed during the course of the disease a total amelioration of the conditions presented. He cites Nonne and Alzheimer as authori-

ties for cases, in which there were symptoms of spinal cord tumors, but which at necropsy showed no anatomic lesions. He notes that Alzheimer attributed the result to faulty methods of examination, but that Alzheimer, contrary to Oppenheim, thought the term "pseudomedullary tumor" should be preserved. They are rare and resemble, clinically, a tumor in the region of the conus medullaris and of the cauda equina. He thinks the adoption of the terms "pseudomedullary, and pseudocerebral, tumors" should only be regarded as provisory, and that they simply express our ignorance of the actual conditions in such cases.

The danger always lies in mistaking the condition, and being under the necessity of making a prognosis of a fatal condition or of mistaking a true tumor for the so-called pseudotype. In the cases presented by the author, different conditions obtain. They are that form of the Brown-Séquard conditoin which is characterized by spontaneous improvement, the latter being their essential attribute.

The final paragraph is concerned with the "therapeutic rationale" of the so-called benign type with which the paper deals. It is difficult to speak of therapy when the etiology is so obscure, and when the recovery is spontaneous. "Specific" treatment is without effect. If improvement follows, it is probably a coincidence. The value of hygienic living cannot be denied, but one patient reported by the author improved under most unfavorable hygienic conditions. Certain patients ascribe their recovery to mineral baths; others to simple baths. With this diversity of lay opinion in view of the spontaneous character of recovery where it occurs, it is needless to say that we are ignorant of what is actually taking place during the course of the disease. The important fact remains, however, that there is a benign form of the Brown-Séquard syndrome.

JONES, Detroit.

LES TUMEURS DIFFUSES DES PAROIS VENTRICULAIRES (NEUROBLASTOMES ENBRYONNAIRES) ETUDE CLINIQUE ET HISTOLOGIQUE. (DIFFUSE TUMORS OF THE VENTRICULAR WALLS—EMBRYONIC NEUROBLASTOMAS. A CLINICAL AND HISTOLOGIC STUDY). E. C. CHRISTIN and F. NAVILLE, *Schweizer Arch. f. Neurol. u. Psychiat.* 7:49, 1920.

The case reported by these authors is exceedingly rare and interesting, both from the clinical and histologic standpoints, so that some of the findings will be discussed somewhat in detail.

The patient was a woman, 54 years of age, with negative family history, who complained of attacks of sudden, intense vertigo and frontal headaches. A bilateral facial paralysis, simultaneous in onset on both sides, appeared rather abruptly. There was progressive general muscular enfeeblement, the sensorium was intermittently clouded, with periods of amnesia; euphoria was at times pronounced. Examination showed a complete paralysis of the left internal and both external recti; the eyes could be turned freely upward and downward. Superficial sensation was slightly diminished over the left trigeminal distribution. The facial muscles were paretic on both sides, more so on the left; no fibrillary tremors were noted. The palate was freely mobile. The cochlear portion of the eighth nerve was normal; the Bárány test was unsatisfactory on account of the ocular paralysis, but as far as it could be carried out, was negative. Both tendo achillis reflexes were abolished. A Wassermann test of the blood was slightly

positive. The diagnosis was uncertain, tumor of the base and myasthenia gravis being considered.

A month later the facial muscles had become completely paralyzed, the patellar reflexes were abolished, there was general asthenia and marked somnolence. A week later there was projectile vomiting and somnolence alternated with delirium. The myasthenic reaction of Jolly was negative. A slight reaction of degeneration was noted over the facial muscles. Fundus examination was negative throughout. The patient was presented before the Medical Society of Geneva, at this time, as an atypical case of myasthenia gravis. The headaches and vomiting continued, as did the progressive cachexia. The spinal fluid contained 26 cells to the c.mm. and 0.2 per cent. albumin. Death occurred about three weeks later.

Necropsy showed some edema of the meninges, slight thickening over the frontal area and a recent hemorrhage near the left first frontal convolution. The exterior of the brain looked normal, and there was no flattening of the convolutions. The arachnoid was somewhat adherent to the right third, sixth and eighth nerves. The ventricular fluid was filled with floating particles. The walls were clothed throughout by a soft, pink, friable, fatty tissue, which the slightest touch sufficed to lacerate. The only interruption of this layer was found at the extreme points of the temporal cornua. It varied from 3 mm. to 1.5 cm. in thickness. The walls of the aqueduct of Sylvius were included, however, the canal was neither obstructed nor dilated; the tumor completely filled the fourth ventricle and even extended a few millimeters down the medullary canal of the cord.

The ependyma, on histologic examination, was found to be almost entirely intact, save here and there where it was completely absent and tumor cells could be found breaking through. The neoplastic tissue itself was made up of delicate fibers, in places closely resembling glia fibers. The blood vessels were everywhere much engorged. Some of the tumor cells resembled lymphocytes; however, there were others which were large and contained large vesicular nuclei and large nucleoli. Numerous mitoses were observed and giant cells were found here and there. The writers were of the opinion that the cells were essentially neuroblastic in type with a certain capacity for differentiating into either ganglion cells or neuroglia cells. They accordingly looked on it as a neuroblastoma.

Tumors of this type are usually found in the suprarenal glands and the sympathetic ganglions. They have been described in the retina. In the nervous system, they are exceedingly rare; in addition to this, the curious distribution found in this case makes it unique. They were able to find six cases reported in the literature, in which involvement of the fourth ventricle alone was the most common. The localization was attributed by Burns to metastases carried in the current of the cerebrospinal fluid. They were reported under the names of sarcoma, embryonic cell glioma approaching syringomyelic gliosis, and ependymal cell glioma.

Tumors of this type have been classified on an embryologic basis by several writers. In the first group are included the neuroblastoma and the sympathoblastoma or ganglioma embryonale sympathicum, made up of undifferentiated nerve or sympathetic cells. In the second group are tumors derived from the chromaffin cells. In the third group is included the ganglioneuroma simplex made up of adult nerve elements with or without myelin, ordinarily benign and appearing in all portions of the body. The last group comprises tumors known

as ganglioneuroblastomas, which includes adult and embryonic elements in varying proportions, appearing either as diffuse growths or as separate nodules.

The writers feel that the ependymal wall does not take place in the formation of these tumors; this opinion, however, is not uniformly accepted. They also think that the tumor proliferation was diffuse from the outset rather than that it started from a limited and isolated center. This conclusion is based on the fact that the ependyma was almost uniformly free from involvement.

Clinically the case presented some interesting features. There was at no time a choked disk. This has been an inconstant finding in tumors of the ventricles, and the writers do not share the opinion of Bollack that choked disk is pathognomonic of dilatation of the third ventricle. They share Reichardt's opinion that it is more likely based on the cerebral reaction to neoplastic irritation. In this case the aqueduct of Sylvius was patent, and there was no evidence of intracranial pressure. The facial paralysis and the paralysis of the external ocular muscles were due in part to neoplastic infiltration and in part to edema. Cerebellar symptoms are frequent, and according to Cimbal, are due to compression, although it may be very slight, on the particularly fragile cells of Purkinje. There is no pathognomonic symptomatology. The symptoms found, however, included progressive apathy, intellectual clouding with amnesia, delirium and agitation, very pronounced muscular asthenia, and progressive amblyopia at times with papilledema and atrophy. On the other hand, there is no impairment of superficial sensibility and no noteworthy involvement of the pyramidal tracts. The diagnosis can never be made with certainty during life. The clinical resemblance to myasthenia gravis is sometimes striking.

WOLTMAN, Rochester, Minn.

ZUR KENNTNIS DES TROUSSEAU'SCHEN PHANOMENS BEI DER TETANIE (INFORMATION PERTAINING TO THE TROUSSEAU PHENOMENON IN TETANY). HARRY SCHÄFFER, *Deutsch. med. Wchnschr.* 46:1073 (Sept. 23) 1920.

The Trousseau sign is considered by the author to be pathognomonic of latent tetany. He discusses his subject from three standpoints.

1. Is the Trousseau sign due to an anemia of limb, or is it due to a stimulation of nerve fibers?

In answering this question the author reviews the work of Schlesinger who shows that the anemia is not the cause of the tetany, but that it is due to stimulation of nerves as demonstrated by the work of Frankl-Hochwart on dogs. Schlesinger showed that in cases of tetany a forced flexion of the thigh in hip, and extension of knee, produced a tetany of the foot in extreme supination, no influence having been exerted on the vascular circulation, the phenomenon being due entirely to a nerve condition. Schlesinger considered that the Trousseau phenomenon would be based on the same condition.

In obtaining the Trousseau sign by means of blood pressure apparatus, it was found that the tetany could be obtained with a pressure slightly less than the diastolic; thus blood pressure—systolic 90, diastolic 60, tetany with 55 mm. of mercury—strong, 45 mm. of mercury—weak, 40 mm. of mercury—none. Several other cases are cited. Occasionally it was found necessary to have the pressure slightly above the diastolic pressure, and it was always possible to feel the radial pulse during such a produced tetany.

The author feels from the array of work done that one is justified in considering the Trousseau sign due to nerve stimulation and not to anemia.

2. Is the Trousseau sign a reflex act?

Increased irritability of motor nerves does not lead to a Trousseau sign. This finding, according to the author, makes the reflex theory possible. Frankl-Hochwart established the reflex quality by producing a bilateral Trousseau sign with constriction of one limb. This was not a constant finding. Another important observation is the fact that the Trousseau sign develops slowly—a summation of stimuli; which would be due more likely to ganglion stimulation than irritation of peripheral nerves, the latter possessing this quality only to a slight degree.

An experimental method for the determination of the nature of the tetany is to be found in a study of the "action current" or potential of the muscle during tetany. According to Piper, voluntary muscular action is characterized by nerve impulse rate which has been demonstrated to be about 50 per second, and called by him rhythm. This rhythm appears to be more marked and distinct as the stimulating impulse is increased. Where this rhythm of 50 is marked, it speaks for the voluntary innervation; that is, for the innervation of the muscle by its anterior horn cells.

If one stimulates the nerve with faradic current, the "action current" curve will show a similar frequency. If the stimulation is kathodal closing tetanus the "action current" curve will show more oscillations, unequal and have a smaller amplitude, and cannot be confused with the curve of a voluntary innervation. During the muscular contraction occurring in a Trousseau phenomenon an "action current" curve of rhythm 50 per second is obtained. For this work a Saitengalvanometer was employed. The author describes the technic and apparatus, illustrating with three cases and several charts.

In the cases, first the voluntary "action current" curve and then the Trousseau "action current" curve were determined. In each case the two curves were practically the same, showing a rhythm of about 50 per second. This work showed that the muscles in a Trousseau phenomenon obtained their impulses in a rhythm of 50, identical to that found in voluntary innervation. This, according to the author, points to an involvement of the central nervous system in the tetany, as such a picture is never obtained by peripheral stimulation.

The Trousseau sign is due to reflex action, and, according to Schäffer, most authorities have come to this conclusion. This agrees with the opinion held by many, namely, that the spinal cord plays a part in tetany. It seems certain that the tetany has its origin in the anterior horn cells and not in the peripheral portion of the neuromuscular apparatus.

3. Even though the Trousseau phenomenon is usually looked on as being tetanic, may it not have a tonic element?

If one observes the thenar and hypothenar eminence, while eliciting a Trousseau sign with gradually applied constriction, one will see a fibrillary contraction of these muscles prior to the tetany. This fibrillation may be kept stationary by the regulation of the pressure, and frequently may constitute the only sign of the phenomenon. Heightened irritability of nervous elements has been suggested and Strümpell called attention to an athetoid movement replacing a Trousseau; that is, a disturbance of the myostatic innervation, the usual form being myomotor. On this ground the Trousseau sign appears as a combination of tonic and tetanic elements in which the former are stimulated by light impulses, as noted in the onset of tetany, greater stimuli producing a reaction in both elements.

An "action current" curve is recorded by the author in a case in which the tetany of the forearm had continued uninterruptedly for six hours. The curve shows a typical fatigue; diminished frequency of oscillation, diminution in height, unevenness in amplitude and increased pauses between isolated oscillations. This curve is similar to that obtained in voluntary muscle fatigue.

The author concludes his article with the following summary:

1. The Trousseau sign may also be obtained in man by direct stimulation of nerve trunks in the median sulcus. Arterial anemia of the arm plays no part in the causation of the tetany.

2. The Trousseau sign may be obtained reflexly; the electromyogram shows a rhythm of 50 oscillations per second in voluntary innervation.

3. The Trousseau phenomenon is a tetany. Most probably the tonic function of the muscle is also involved.

4. The "action current" curve of muscles following prolonged tetany simulates that of voluntary innervated muscle in severe fatigue.

MOERSCH, Rochester, Minn.

THE MECHANISM OF INVOLUTIONARY MELANCHOLIA. W. F. MENZIES, *J. Men. Sc.* **66**:355 (Oct.) 1920.

We have in the presidential address to the Medico-Psychological Association of Great Britain a most unusual effort by Dr. Menzies. It is a curious combination of definite detail and the wide reaches of the indefinite. About half of the paper is concerned with data about the anatomy, physiology and chemistry of the conduction of the nervous impulse, disclosing a wealth of information: the other half attempts to connect these facts with melancholia, as known to psychiatrists. While there are many statements which arouse instant criticism and even denial, it is perhaps fair to think that the address as a whole is offered as suggestive rather than argumentative material.

Nowhere in the article can be found a definition either of "involutionary" or of "melancholia." We may infer that the author is considering any state of depression, quiet or active, occurring in the down grade of life. "The choice of involutionary melancholia was to eliminate the positive influence of thymus, thyroid and ovaries, which in presenility are more or less atrophic." So the title is not to be read as our term "involution melancholia."

The author states that the psychology of melancholia is the depressive emotion, and the anatomy of the depressive emotion is a sympatheticotonus, and the chemistry of a sympatheticotonus is an endogenous anoxemia. As a most important factor is excessive putrefaction in the intestine, a discussion of Lane's inclusive list of symptoms of enterostasis and of bacterial absorption follows. To understand (page 359) the connection between bacterial absorption and melancholia we need an anatomic basis, and this is found in the involuntary nervous system, whose embryology and physiology are sketched. The next step is to show that many of the phenomena which are produced by emotion and also by nervous outflow from the involuntary system are closely duplicated by the effects of epinephrin. "An emotional outflow is at one with the sympatheticotonus, and is accompanied by an increase of adrenalin" which is free by a complicated process to liberate from the liver an excess of glucose. Glycosuria is comparatively common in melancholia, and the amount of sugar in the urine varies with the amount of the depression (Raimann, Schultze, Goodhart, Bond, Toy, Knauer). Kooy in blood sugar percentages finds that melancholiac patients are above nor-

mal people and other mental patients: Reimann found lessened sugar tolerance in both excitements and depressions. Also "it would appear that in melancholia there is a chronic hyperadrenalism."

The author's description of the symptoms of states of depression is given rather fully, because much exception can be taken to its details. The heart is weak and the pulse frequent, soft. The systolic blood pressure in the erect posture is low. This statement seems remarkable to the reviewer in view of the statements in the article that when blood pressure was high, it was found to be in a case of chronic kidney disease; that atheroma occurs in almost every case of involutionary melancholia; that most such patients suffer from renal cirrhosis. Again, in melancholia the skin and viscera are poorly supplied with blood, as are the secretory glands. There is constipation and checking of the "normal harmonic train of digestion," especially at the pylorus. Accommodation is impaired and the pupils are large. In some patients a "proximal rigidity" (Stoddart) is shown.

Suprarenal extract, in comparison, relaxes smooth bronchiole muscle, counteracts fatigue, draws blood from the viscera to the lungs, the central nervous system and skeletal muscles, hastens coagulation and increases the sugar release from the liver.

So the author concludes that the state of depression is nothing more than a persisting sympatheticotonus, to which are added two complications sufficiently inclusive—bacterial toxemia from enterostasis and unconscious memory.

In putting forward the theory that in states of depression there is general anoxemia, the statement is quoted (Townsend and others) that in melancholia there is an increased excretion of potassium phenylsulphate and indoxylsulphate, and in the blood stream an undue amount of cholesterolin, lecithin, phosphates and calcium.

It would be cause for the greatest congratulation if the contents of the blood stream in melancholia were as definitely known as the author implies.

Summarizing the views in this closely packed address: Involution and melancholia are concomitant, depending on different modifications of the fight by liver antitoxins against intestinal toxins, "the penalty of failure being deficient oxidation." In response to suboxidation appears a sympatheticotonus, the sensorimotor resultant of which is the depressive emotion. The supragranular layers of the cortex are so affected by lack of oxygen that association is diminished, and the infragranular layers allow unconscious memories to slip from under control.

BOND, Philadelphia.

DIE HISTOPATHOLOGISCHE ZUSAMMENGEHÖRIGKEIT DER WILSONSCHEN KRANKHEIT UND DER PSEUDOSCLEROSE (HISTOPATHOLOGIC IDENTITY OF WILSON'S DISEASE AND PSEUDOSCLEROSIS). W. SPIELMEYER, *Ztschr. f. d. ges. Neurol. u. Psychiat.* 57:312, 1920.

Spielmeyer refers to a number of recent cases in which the similarity of the clinical picture between Wilson's disease and pseudosclerosis has led neurologists to believe that these two separately described affections are fundamentally the same. The writer has approached this problem from the histopathologic standpoint. The material for study consisted of seven cases: two personal clinical cases; three from Nissl's collection clinically diagnosed as lenticular degeneration with liver sclerosis and so verified by necropsy; one case of pseudosclerosis described by Fleischer and microscopically examined by Brodmann; and, finally, the original classic case of Hösslin-Alzheimer, which served as a control.

The writer recalls that Wilson's disease from the standpoint of brain pathology is held to be an elective degenerative process of the corpus striatum, the cerebral cortex showing no noteworthy changes. Alzheimer finds in pseudosclerosis (Westphal-Strümpell) the principal lesions in the corpus striatum, optic thalamus, subthalamic region, pons and dentate nucleus of the cerebellum, but lesions also occur in other parts of the brain and especially in the cerebral cortex. The essential pathologic picture is the occurrence of the giant glia cells with large lobulated nuclei poor in chromatin and with small cell bodies. They show no tendency to fiber formation and ultimately degenerate.

Fleischner's case corresponded in practically all particulars with Alzheimer's case of pseudosclerosis. In the personal cases of the author, in which the clinical differentiation was impossible, Case E, in which the patient died at the age of 17 years, after an illness of 2½ years, belonged to the group of those suffering from liver cirrhosis. Macroscopically this case was remarkable because of absence of softening or atrophy of the lenticular nucleus. Microscopically, in the corpus striatum a poverty of ganglion cells and increase of small glia cells without fiber formation was noted, also the presence of lattice cells and granule cells. The mesenchymal network was increased, but not the blood vessels. The dentate nucleus revealed degenerated nerve cells and pathologic neuroglia. Nowhere was there evidence of an inflammatory process or of vascular degeneration.

The disease in the other patient (Case H), who died at 15 years of age, ran a course of eight years. The liver was normal. There was a striking atrophy of the caudate nucleus and of all parts of the corpus striatum but most marked in the putamen. A true softening cystic degeneration or cavity formation was not present; only in the outer third of the lenticular nucleus were widened peri-adventitial spaces seen about the vessels. This nucleus presented a considerable increase in glia fibers. The absence of cellular disintegration or presence of any considerable evidence of scavenger cells seemed to indicate that the abnormal process was either arrested or slowly progressing. No changes were present in the dentate nucleus, and the characteristic Alzheimer glia cells were here absent. In the cerebral cortex, however, a most unusual ganglion cell degeneration was found—a pale rounded, often pear-shaped, cell body with broken up nuclear chromatin content. Rod cells and neurophagic glia accompanied the process.

The three cases of typical lenticular degeneration were all characterized by cystic degeneration or marked tissue destruction in the lenticular nucleus, but in all cases the Alzheimer glia cells were found. Glial fiber increase and granule cells from the neuroglia were more or less constant features. Of great significance in these cases was an increase in the smaller blood vessels and a mesenchymal adventitial increase between the blood vessels. The most important fact, however, was the discovery that the pathologic process in Wilson's disease is not so sharply localized as is believed. In these cases changes were found in the cerebral cortex, both of the cell type of Alzheimer and also cystic degeneration resembling that in the lenticular nucleus itself. The dentate nucleus was also found involved.

The author believes that the same disease process is common to both Wilson's disease and to pseudosclerosis and that a differentiation on histopathologic grounds is not possible. He does not believe that either disease is properly speaking a system disease. The writer discusses the nature of the unusual histologic reactions. The article is well illustrated with microscopic reproductions.

SCHALLER, San Francisco

UEBER DIE BEEINFLUSSUNG VON VISIONEN DURCH CEREBELLAR AUSAELOSTE VESTIBULARE UND OPHTHALMOSTATISCHE STORUNGEN (VESTIBULAR AND OPHTHALMOSTATIC INFLUENCES ON VISION [OF HALLUCINATORY NATURE]). A. Pick, *Ztschr. f. d. ges. Neurol. u. Psychiat.* 56:213, 1920.

Pick believes that he can show that a series of events which hitherto have been believed to be hallucinations are not of this character but are caused by disturbances originating from the vestibular and ophthalmostatic apparatus. He cites at great length and with minute details the case of an engineer of 61 with a diagnosis of tabes and arteriosclerosis, who was subject to hallucinations in which he in his bed would seem to travel through the walls of his rooms and see double the various phenomena which appeared before him somewhat in the nature of a film with a negative and a positive exposure. The account of the patient's subjective statements regarding his hallucinations must be read in the original to be appreciated. The stories vary from his ability during these attacks to look through the roof, through the walls, to see objects beyond the room as doubled to peculiar contortions, of objects bending forward, contortion of the ceiling, etc.

Necropsy examination revealed a minor degree of atrophy of the frontal lobes, multiple pea-shaped, slightly shrunken brown spots in the nuclei lenticularis and in the dentate nuclei of both hemispheres of the cerebellum. What seemed to be a pigmented scar, microscopic examination showed to be a peculiar glial degeneration.

In the most careful physical examination there is, however, the glaring omission of an examination of the vestibular apparatus, and in the absence of this the reviewer does not see how any conclusion regarding the effect of cerebellar disturbances affecting the vestibular apparatus and having any influence on the double vision and slanting or broken outlines of objects can be drawn.

As the conclusions depended on the statements of the patient, the effect of suggestion by constant questioning cannot be eliminated. Through the entire history the fact that constant questions tending to illustrate whether he saw double, whether the unreal objects or the real objects—both of which appeared in hallucinations—were seen double, all impresses one with the great possibility of his answers being made to conform unconsciously to suggestions given in questions.

The author, in reviewing the voluminous statements of the patient during a long talk, decides that the patient really never saw any visions double, and he always denied having seen them double except in one instance. The author concludes that one is always dealing with cases of hysteria when visions are reported as having been double.

Pick states that we are well aware of similar cases in delirium tremens and know the extraordinary power of suggestion on the character of the hallucinations and visions of the patients.

In the voluminous literature no observation has been made of any cases of visions seen as double in spite of the fact that among these patients were many with an ocular palsy. It would seem quite natural that this should not be so and that there is no connection at all between the two affairs. One is a purely motor and muscular affair, the other a psychic disturbance, and they bear no relation to each other.

He ends the article by drawing attention to the observations of P. Schelder, which are really of great importance and interest. He noticed that a straight line was made to seem to bend forward by cold water irritation of one labyrinth and that it seemed to bend in the opposite direction when this irritation ceased. With the appearance of nystagmus this line seemed to be chopped up into several pieces separated from each other in the horizontal meridian.

In the absence of any vestibular examination in the case that Pick mentions, the importance of the deductions he makes is questionable.

BARKAN, San Francisco.

DIE KLINISCHE BEDEUTUNG DES FACIALS PHAENOMENS IM SAEUGLING- UND KINDESALTER (THE CLINICAL SIGNIFICANCE OF THE FACIAL REFLEX (CHVOSTEK SIGN) IN INFANCY AND IN CHILDHOOD). K. BLÜHDORN, *Med. Klin.* **16**:1009 (Oct. 24) 1920.

According to Blühdorn, spasmophilia occurs most frequently during the first two years of life. It may, however, occur later, if only as a latent tendency. Attacks are most common in the late winter and in the early spring. Heightened mechanical and electrical excitability are characteristic findings in spasmophilia, while almost pathognomonic is the kathodal opening contraction reaction obtained by stimulation of the median nerve with a current of 5 milliamperes or less. In older children 6 milliamperes should be used as the upper limit of current employed. A reversal of the formula in which anodal opening contraction is greater than anodal closure contraction is also a fairly constant finding. The appearance of kathodal closing tetanus with the above amount of current is pathologic.

As electrical apparatus is not always at hand, it is desirable to avail oneself of any other test which may prove satisfactory. According to the author, the Trousseau sign, while usually positive in spasmophilia, is not as constant as the Chvostek. Some authorities consider the Chvostek a sign of a neuropathic constitution, while others doubt its value. The author believes it to be a constant finding and not varying from infancy to childhood.

In children with rickets or showing other signs of a possible spasmophilia, a positive Chvostek sign and a heightened electrical excitability was obtained in the majority of cases, the Chvostek sign being the more constant. Finkelstein found evidence of latent spasmophilia in 55 per cent. of a group of artificially fed children. It is the author's opinion that latent spasmophilia does not necessarily mean a true spasmophilia; the possibility should, however, be recognized and the parents instructed in the proper care of the child. In an examination of thirty-seven latent spasmophilic children 81 per cent. gave a positive Chvostek sign. According to Blühdorn, the facial reflex or Chvostek sign, as compared with the electrical reactions, is the more lasting.

During, or directly following, a spasmophilic attack, the Chvostek sign may be absent. What the relation may be between the facial reflex and the electrical reactions is still a question in the opinion of the author.

In the review of the author's material, neuropathic tendencies constantly presented themselves, not only in the patient but also in the family. It is his opinion that spasmophilic persons are neuropathic, but that not all neuropathic persons harbor a latent spasmophilia. Spasmophilia was frequently found associated with other nervous ailments, meningitis and encephalitis being mentioned by the author.

The presence of a positive facial reflex in epilepsy is not uncommon and brings up the question of the relation of spasmophilia to epilepsy. It is not uncommon for a spasmophilic person to develop petit mal attacks or attacks resembling them, in which luminal does no good. Narcolepsy also has been observed as a sequel of spasmophilia. In the latter cases a positive Chvostek sign was present, and good results were obtained with calcium therapy. Spasmophilic patients showing intelligence defects and typical epileptic attacks must be considered as having true epilepsy, and are said by Potpetschnigg to have tetanoid epilepsy.

Teething cramps, whooping cough or convulsions at the onset of fever are indicative of latent spasmophilia, and such patients as a rule show a positive Chvostek sign. In obtaining a history of the patient, it is frequently impossible to get this information as the patient is unaware of the fact and apparently the parents have forgotten the occurrence.

In conclusion, the author discusses the therapy of spasmophilia in which calcium forms the chief factor. Of interest is the fact that in nervous children in whom there is a latent spasmophilia as noted by the Chvostek sign and the electrical reactions, good results are obtained by calcium medication.

MOERSCH, Rochester, Minn.

DISMORFIE CRANICHE DA APLASIA EMICEREBELLARE (CRANIAL DEFORMITIES FROM HEMICEREBELLAR APLASIA). G. MINGAZZINI and F. GIANNULI, *Riv. di Antropol.* 22:33, 1918.

Mingazzini and Giannuli, starting from an anatomic study of the brain and skull of a case of fat eunuchoid with hemicerbellar aplasia, have come to some important conclusions on the relationship existing between the morphology of the skull and that of the brain.

Among the many anomalies which the skull of this patient showed, it was found that the opening of the transverse sinus in the left jugular vein was almost obliterated by an anomalous bone formation; and that the anterior, the middle and the posterior clinoid processes were united to one another by osseous bridges which formed three foramina, the anterior foramen opticum, the middle foramen clino-carotideum and the posterior foramen clino-clinoideum. The sella turcica was deep and very narrow, and the marked hyperostosis of the tuberculum ephippii had caused the peduncle of the hypophysis to be choked, a condition which led to the clinical picture of eunuchoidism.

As to the possible pathogenesis of the hyperostosis, the authors, in consideration of the marked thickening of the diploe, particularly that of the right frontal region such as is found frequently in syphilitic patients, think that in this case hereditary syphilis may have been responsible both for the osseous alterations and for the cerebellar aplasia, the latter probably being the consequence of a specific arteritic process of one of the inferior cerebellar arteries and of a branch of the superior cerebellar artery. Probably such syphilitic processes took place during prenatal life, as shown by the fetal characteristics of some internal formations of the occipital bone and by certain features disclosed in the study of the brain.

Another important feature of this skull is the absence of harmonious correlation between its internal and its external morphology. The volumetric valuation of the different cranial fossae, and the study of the serial cuts of the brain indicated that the compression exerted by the left occipital lobe from behind on the middle line toward the right side, had caused deformation of the whole.

right cerebral hemisphere. In fact, the right *girus hippocampi* and the *girus fusiformis* had been forced to a vertical position between the pulvinar and the lateral ventricle, thus preventing the formation of the retrolenticular segment of the internal capsule.

Compensation on the external formation of the bones so well concealed the disharmonies of the inner surface of the skull cap as to give to this skull, when examined from the outside, a most harmonious morphologic aspect correspondent to the ethnic type of the species. This confirms the fact that the value of the external cranic form is less important than the value of the internal one.

Here the authors report extensively on the anthropometric measurements and give a minute description of the deformities found in the cranial cavity. They give a confirmation to the views expressed by Tedeschi in his craniologic studies, saying that "not always do disturbances of development of portions of the brains, correspond to, in relation of reciprocal dependence, analogous alterations in the corresponding portions of the skull-cap." In view of the tendency shown by Schwalbe to follow the old conceptions of Gall and Moebius, in an attempt to read on the external surface of the skull the sulci of the convolutions, the authors warn against relying too much on the external morphology of the skull. Without denying that such an attempt may lead to some practical results for the cranial surgery, they entertain no hope that the same may be said from the anthropologic point of view, since the fundamental questions regarding the relations existing between the development of the brain and the development of the skull still remain on the solid bases laid by Broca, Gudden, and Virchow.

NACCARATI, New York.

ZUR FRAGE DER ROLLE DES RUCKENMARKS BEI EPILEPTISCHEN KRAMPFEN (THE ROLE OF THE SPINAL CORD IN EPILEPTIFORM CONVULSIONS). M. LAPINSKY, *Neurol. Centralbl.* **39**:324, 1920.

The author discusses the rôle played by the spinal cord in epileptiform attacks and cites cases of injury or tumors of the spinal cord with convulsive attacks. Brown-Séquard produced the epileptiform attacks in a guinea-pig by stimulation of the spinal cord when the cerebellum, the pons and the medulla were removed. Marsh attempted to repeat these experiments on frogs, but failed. With creatin, Laudois produced some irregular contractions but no regular convulsions, and concluded that the spinal cord plays a passive and conductive rôle in convulsions due to cortical irritation.

The author attempts by experiments to answer these questions: 1. Can one produce epileptiform attacks in frogs by irritation of the spinal cord? 2. Over which spinal tracts may convulsive impulses travel that arise in the cerebral hemispheres or in the medulla?

The first query was studied by stimulation of the lower part of the spinal cord in frogs with creatin, salt and the faradic current after its section at the level of the second dorsal vertebra. In this experiment he was unable to produce any convulsive attacks. The second problem was investigated by making partial section of the cord at the third and fourth dorsal vertebra. Five series of animals were thus treated, the undivided bridge consisting respectively of the anterior, posterior, both lateral columns, one lateral column and one-half of the cord; variable degrees of paralysis and anesthesia in the hind legs resulted.

Convulsive attacks were induced by application of creatin to the cerebral cortex or medulla. The reflex excitability of the cord was elicited by concentrated acetic acid applied to the lower extremities.

From the experimental study, the following conclusions were drawn: First, during convulsions of cortical or bulbar origin that impulse can be transmitted, not only lengthwise of the cord, but also transversely. The longitudinal conduction is accomplished by the anterior and lateral column, and the transverse conduction of impulses from one half of the cord to the other occurs apparently through the gray substance. The transmission of the convulsions through the posterior extremities was not possible, while the transverse conduction was greatly reduced. Second, in all cases in which the upper and lower sections of the cord were joined by a small bridge, the posterior extremities were partially or completely paralyzed; voluntary movements were impossible, but nevertheless, epileptiform convulsions were able to pass. The reflexes in the posterior extremities were either lessened or normal. Third, as the convulsions in the hind legs could be interrupted by stimulation of these parts, while the attack continued its usual course in the fore part of the body, the convulsive excitation of the hind legs was weak, probably because the diameter of the bridge which conducted the impulse was too narrow and the impulse was too slight. Fourth, the insufficient irritation discharge in the posterior part of the spinal cord during the convulsive discharge in the higher centers causes these sections to remain less exhausted, and the reflex excitability to exist during the convulsion. Fifth, the spinal cord cannot originate the epileptiform discharge, but serves only to transmit the impulses which arise in the brain and medulla.

SHELDEN, Rochester, Minn.

A PROPOS DE NEURO-FIBROMATOSES CENTRALES. (CONCERNING CENTRAL NEUROFIBROMATOSIS). E. CHRISTIN and F. NAVILLE; *Ann. de méd.* 8:30 (July) 1920.

The authors review the literature of twenty recorded cases and have four well studied cases of their own to add, one with detailed histologic reports.

Generalized central neurofibromatosis is characterized by the presence of multiple neoformations located on the course of the cranial or spinal nerves, or within the medulla, brain proper or meninges.

The histologic structure of these tumors is varied. All the tumors which neuroglia and mesodermic perineural tissues can furnish are represented. Even in a single person, such as in their first case, there may be glioma, fibroma, true neuroma, myxoma, osteoma, endothelioma and sarcoma. The process seems to amount to a teratoid disposition on the part of the tissues. This impression is reinforced by the frequent concomitant occurrence of diverse congenital malformations and even of teratoid tumors properly speaking.

The eighth nerve is almost always involved and usually bilaterally. Among the other nerves, the fourth is the most often affected, and then follow the seventh and ninth and then the third and fifth. The other nerves are rarely involved. These statements relate to the intracranial course of the nerves for, extracranially considered, the fifth and tenth are most often affected.

In sixteen cases in which the author has exact data, the first symptoms appeared prior to the age of 20.

It is necessary to bear in mind the long latent phase which may occur. In one case, deafness was the only symptom over a period of seventeen years, and the only symptom during twenty-five years in another.

Lesions of the acoustic nerves are as signatures of the disease. There was bilateral central deafness in sixteen of twenty-three cases, unilateral in four. Vestibular symptoms are frequent, but not so striking. Equilibrium, even in tests with the revolving chair, was little disturbed in all the cases in spite of the abolition of the labyrinthian reactions to the galvanic current and to water.

This apparent contradictory symptomatology merits exact research. Another feature likewise requiring explanation is the frequent absence of headaches, torpor or somnolence in spite of the size and extraordinary number of tumors present intracranially in some cases (for example, in the authors' case).

There has been a combination of cutaneous neurofibromas with intracranial localization of the process in less than 40 per cent of the cases.

DAVIS, New York.

REPORTS ON PSYCHIATRY. HENRY R. STEDMAN and DONALD J. MACPHERSON, Boston M. & S. J. **183**:579 (Nov. 11) 1920.

Stedman and Macpherson present a résumé of some of the more important and recent psychiatric achievements. Ball,¹ Southard,² Cobb,³ and others have made contributions to our knowledge of psychiatry in its relationship to industrial efficiency. It is interesting to note that a neuropsychiatric survey of fifty-seven employees of an industrial company shortly before a strike showed considerable nervous and mental abnormality and in every instance a psychopathic history. It is an established fact that the preliminary neuropsychiatric examination of recruits was one of the important factors in the production of an efficient army. Industry should not disregard this important lesson of the great war. Two steps which are immediately possible in this connection are: first, the establishment of medicopsychologic laboratories in the employment bureaus of the more important and larger industrial concerns, and, second, the teaching of the principles of psychiatry and psychology to industrial physicians.

Campbell,⁴ in a recent paper, analyzed the influence of poor mental hygiene during childhood. Parents and teachers who should be the instruments for the greatest good in this direction are too often ignorant of even elementary child psychology. Thorough instruction in the problems of maladjustment is advocated, especially for the university and the medical student.

Rossi⁵ makes a distinct addition to our knowledge of endocrine disorders in the psychoses by his report of nine cases of manic-depressive insanity in which he was able to demonstrate suprarenal insufficiency and produced a favorable therapeutic influence by the exhibition of epinephrin.

Lafora,⁶ in presenting fifteen cases of paresis and eight of tabes showing marked improvement under treatment, particularly emphasizes the necessity of early diagnosis. The author states that general paralysis can often be detected before the appearance of clinical symptoms by the application of the Wassermann reaction to 0.2 c.c. of the spinal fluid and the colloidal gold test. Lafora uses the autoserum prepared in vitro by the Byrnes and Ogilvie methods. Four

1. Ball: *Am. J. Insan.* **75**:521 (April) 1919.

2. Southard: *Industrial Management*, February, 1920.

3. Cobb: *J. Industrial Hygiene* **1**:343 (Nov.) 1919.

4. Campbell: *Mental Hygiene* **3**:199 (April) 1919.

5. Rossi: *An. de la Fac. de med., Montevideo* **4**:358 (Dec.) 1919.

6. Safora: *Rev. neurol.* **35**:625 (Aug.) 1919.

or five injections are made by the vein at intervals of from four to six days, 0.01 grain of mercury and arsphenamin. The first intraspinal injection was made with arsphenamized serum, never more than 2 or 3 mg., and the maximal dose ever reached was 4 mg. of mercuric chlorid or 7 mg. of (French) neo-arsphenamin. The intervals were from twenty to forty days, while the injections by the vein were made about twice a week alternating the mercury with the neo-arsphenamin and suspending for two or three weeks at the end of six months.

There were two contributions by neurohistologists: C. von Monakow and S. Kitaboyashasi⁷ demonstrated choroid plexus abnormalities in twelve cases of dementia praecox, including hyperemia, scattered degeneration of the villi, amyloid degeneration of the connective tissue, collection of colloid masses within the plexus and interpapillary exudation. From Mott's⁸ laboratory comes a study of the testicle in various psychoses with a demonstration of primary testicular atrophy peculiar to dementia praecox.

STRECKER, Philadelphia.

BEITRAG ZUR KENNTNIS DER RUECKBILDUNG VON APRAXIE
(CONTRIBUTION TO THE STUDY OF THE RESOLUTION OF
AN APRAXIA). HANS SEELERT, *Monatschr. f. Psychiat. & Neurol.* **48**:
125 (Sept.) 1920.

The author reports a case of carbon monoxid poisoning, in which the general symptoms of cerebral disturbance were accompanied by aphasia and apraxia. Evidence of involvement of motor and sensory paths was slight, and disappeared early. Some disorientation in space was present at first but cleared while the apraxia was still at its height. The case was especially favorable for study because the general psychic and aphasic manifestations cleared up early leaving an uncomplicated apraxia.

The apraxia was of the ideatory type. It was manifested in a gross disturbance in the ability to use objects properly, to make purposive movements and to carry out motions of expression; writing and drawing were impossible. As the condition improved, the ability to write returned. Gradually he regained the ability to use objects properly, to make purposive movements and to carry out motions of expression. Even after this improvement had advanced quite far, he had great difficulty in initiating such movements. The ability to draw, both from memory and by copying, improved last.

In the end everything cleared except his ability to copy. The psychic disturbance had entirely disappeared, so that it could be demonstrated that the difficulty in copying was due to apraxia alone and not to any failure of optical understanding; the patient could recognize and name the object to be copied, could criticize and in a measure correct his unsuccessful attempts, and could hold in memory geometrical figures so as to be able to pick them out of a group. In other words, the afferent optic paths were normal, and a normal optic engram could be formed and retained. The difficulty came in converting the optic engram into the motor act of drawing.

To copy normally, the optic picture must be normal; the sensorimotor functions of the hand must be normal; and the association paths between the optic center and the hand center must be intact. The author believes that in this case the association paths and the sensorimotor area were both involved.

7. Von Monakow, C., and Kitaboyashasi, S.: *Schweiz. Arch. f. Neurol. u. Psychiat.* **4**:13, 1919.

8. Mott: *Brit. M. J.* **2**:655 (Nov. 22) 1919.

As to the anatomic localization, this case would seem to confirm the view that ideatory apraxia is dependent on a diffuse disturbance of the brain.

SELLING, Portland, Ore.

INTRASPINAL TREATMENT IN NEUROSYPHILIS. J. H. MITCHELL.
Arch. Dermat. & Syph. 2:44 (July) 1920.

The author briefly reviews the literature on this subject and reports his own technic and the results he has obtained.

The patients were given an intravenous injection of arsphenamin in the early afternoon, and from fifteen to forty minutes later 100 c.c. of blood were withdrawn. The blood was kept in an icebox, after clotting had occurred, until the next morning. The serum was then decanted, centrifugated and inactivated in the usual manner. After withdrawing about 30 to 35 c.c. of spinal fluid, a like amount of serum was injected. Reactions were not severe and morphia, hypodermatically injected, was sufficient to control the pain. The patients were able to leave the hospital usually by noon of the following day. The efficacy of the therapy was constantly watched and followed by serologic examinations after each injection.

The author employs this method only when patients show evidences of neurosyphilis and do not respond to the intravenous medication. He believes that by this technic many cases can be reached that show persistence of signs and symptoms after fair trial of intravenous medication. As a rule, the intraspinal treatments were given once a month and a course of eight to ten intravenous treatments preceded the intraspinal. In all, fifty-two patients were treated. Statistical study of the twenty patients with paresis treated showed that those who received the greater number of treatments made the best progress; of the twenty-five cases of tabes, thirteen patients improved clinically and have a negative spinal fluid, ten improved clinically but have a positive spinal fluid; of seven cases of syphilis of the nervous system, four patients improved clinically and reached negative fluids, while one case showed a fluid which became negative then returned to positive, and another remained strongly positive.

The paper is interesting from a comparative study of the different methods of treatment of nervous system syphilis; however, no evidence is adduced to show that this modification of the Swift-Ellis treatment, as applied by the author, has any advantages over the various other methods and modifications.

PATTEN, Philadelphia.

THE PIGMENTARY, GROWTH AND ENDOCRINE DISTURBANCES
INDUCED IN THE ANURAN TADPOLE BY THE EARLY ABLATION OF THE PARS BUCCALIS OF THE HYPOPHYSIS. P. E. SMITH, Am. Anatomical Memoirs, No. 11 (Oct.) 1920.

This important paper contributes significant data regarding the puzzling questions relating to the functional interrelationships of the different endocrine organs. The anterior part (pars glandularis) of the hypophysis can be completely removed with a minimum of disturbance to other tissues in tadpoles of *Rana boylei* from 3.5 to 4 mm. long. The animals survive the operation and grow well, thus affording opportunity for various observations and nutritional and pharmacologic experiments.

The hypophysectomized animals are albinos, and the pigmentary changes are carefully analyzed. These albinos exhibit a retardation of growth and do not

metamorphose. It is shown that in the normal animals the growth-maintaining substance is derived from the anterior lobe, and if tadpoles deprived of this lobe are fed on the fresh anterior lobe of the beef they exhibit a nearly normal rate of growth. Since these animals do not metamorphose, their growth extends beyond the normal larval period and, accordingly, they ultimately attain a size in excess of the normal. Extraction of the anterior lobe substance with neither boiling water nor boiling absolute alcohol removes the growth-maintaining substance, but the residues remaining after such extraction, if fed to hypophysectomized larvae, bring their growth rates up to normal.

In larvae deprived of the anterior lobe of the hypophysis all other glands of internal secretion are reduced in size, with the exception of the suprarenal medulla, which is structurally altered and may be slightly enlarged. Early removal of the thyroids, on the other hand, results in enlargement of all other glands of internal secretion, again excepting the suprarenal medulla.

After removal of the anterior lobe of the hypophysis, the posterior lobe is diminished in size, asymmetrical, and atypical in position. Moreover, in these larvae the floor of the infundibulum, normally in contact with the anterior lobe, does not thicken in later development, as does the normal, but remains thin and membranous. These points are indicative of an intimate physiologic relationship between the so-called glandular and nervous parts of the hypophysis.

HERRICK, Chicago.

ACUTE CEREBROCEREBELLAR ATAXIA (WITH REPORT OF CASES). J. P. CROZER GRIFFITH, *Am. J. Dis. Child.* **20**:82 (Aug.) 1920.

Cerebrocerebellar ataxia is occasionally observed as a manifestation of lethargic encephalitis. Four cases are reported with observations and comments. The first case followed influenza and presented the symptoms of ataxic movements, nystagmus and disordered speech. The second case did not follow influenza but was ushered in by weakness in the left hand, dizziness, staggering to the left when walking, or falling to the left (sometimes to the right), and ataxic movements on the left, all of which improved after a time. On examination, four years after the initial symptoms, the patient showed some ataxia persisting in the left hand and tremors. The third case followed an attack of measles, beginning with internal strabismus, difficulty in talking, dizziness and mental apathy. Later there were nystagmus (constant), tendency to go to the left in walking, or falling to the left, and a Bárány test located a lesion in the upper posterior portion of the pons and anterior superior portion of the adjacent cerebellum; gradual improvement took place, but some months later the patient returned to the hospital with an exaggeration of all symptoms, and with the interim history that the nystagmus and strabismus had persisted. The fourth case possibly followed an influenzal attack. The encephalitis began with vomiting, constipation, stupor and high temperature, followed by convulsive seizures of short duration, coming on at intervals over a period of sixty hours. The coma persisted for several days, and speech did not return for four months or longer. A year after the attack ataxia in movement and slow speech persisted.

The author draws the conclusions that an acute hemorrhagic encephalitis involves the cerebellum more frequently than is often suspected, and when both cerebrum and cerebellum are affected the term "cerebrocerebellar encephalitis" should be preferred. The cause seems to be some infectious disease and

the usual symptoms are: unconsciousness, ataxia, affection of speech, disturbance of mentality, nystagmus, loss of power (sometimes) and persistence of reflexes. As to recovery, he concludes from his own experiences and the reports of others that in the majority of instances recovery will occur, but that it is often delayed and slow.

PATTEN, Philadelphia.

NERVE OVERLAP AS RELATED TO THE RELATIVELY EARLY RETURN OF PAIN SENSE FOLLOWING INJURY TO THE PERIPHERAL NERVES. LEWIS J. POLLOCK, *J. Comp. Neurol.* **32**:357 (Dec. 15) 1920.

Pollock has previously reported on 1,020 cases of peripheral nerve lesion observed in American military hospitals.¹ In this shorter communication 132 of these cases, which were controlled by operation, are analyzed with exclusive reference to the question of the relation of the phenomena of pain sensibility in areas of nerve overlap to the protopathic type of sensibility as described by Head and his colleagues. He finds that the dissociated return of sensibility of cutaneous pain occurs only in areas of possible overlap. When several nerves serving adjacent areas are severed simultaneously, sensibility to cutaneous pain is not present after injury, nor does it return before tactile sensibility in the borders between these areas where it is usually present when either nerve is severed alone. When a region in the area of sensory distribution of a severed nerve is sensitive to cutaneous pain, and the region is adjacent to another nerve area, if this nerve is severed, complete analgesia results in the previously sensitive region. When sensibility to prick pain is present or returns in the area of possible overlap on to the sensory distribution of the severed nerve, subsequent resection and suture of this nerve does not change the general extent of this sensitive area, although the borders may at times be slightly enlarged or diminished; that is, the pain sense returned or present before the operation was not due to partial regeneration. Because of these observations, it is believed that the early and dissociated return of pain sense attributed by Head to the lawless regeneration of protopathic sensibility is due to the assumption of function of adjacent and overlapping nerves.

HERRICK, Chicago

SYMPTOMATOLOGY OF SPINAL CORD TUMORS. ISADOR ABRAHAMSON and HYMAN CLIMENKO, *J. A. M. A.* **75**:1126 (Oct. 23) 1920.

All cases of transverse myelitis with progressive symptoms should be regarded with suspicion and thoroughly studied, particularly as to level signs. The symptoms and signs of soft intramedullary growths are somewhat different from the hard tumors, the motor and sensory symptoms showing considerable parallelism in contrast to the stationary and earlier motor symptoms of the hard tumors. Xanthochromia is more common in soft tumors, and root symptoms more frequently are bilateral. Pain is present in all soft intramedullary tumors that reach the posterior roots, or the surface of the cord, but this may be a late happening, while in extramedullary growths the symptoms develop more rapidly. Tract sensory signs and trophic changes are more common in intramedullary growth, but rectal and vesical signs are less frequent.

1. Pollock, L. J.: Overlap of So-Called Protopathic Sensibility as Seen in Peripheral Nerve Lesions, *Arch. Neurol. & Psychiat.* **2**:667 (Dec.) 1919.

When no distinct level can be established, the effect of lumbar puncture should be carefully watched and operation only undertaken when a definite level is determined. The possibility of soft tumors in atypical cord lesions must be kept in mind. For determination of actual site of tumors certain signs have proved to be of value: (a) root signs, which consist of neuralgic pains with symptoms pointing to a distal distribution of an affected nerve; (b) zones of hyperesthesia immediately above the seat of the tumor and more marked at the homolateral site; (c) level abolition of skin and tendon reflexes; (d) deep spinal tenderness; (e) alteration in vibratory sense; (f) no sweating below the level after injection; (g) ocular symptoms, such as lateral nystagmus and difference in the pupillary size, as well as palpebral fissure, which may all be present in high cervical tumors. The less reliable signs are: (a) homolateral paralysis; (b) heterolateral distribution of pain and temperature sense which reach their highest level only after considerable cord compression, late in the disease; (c) this sensory loss when complete is usually three segments below the actual level of the tumor.

Operations, frequently by their decompressive effects, help even in intra-medullary tumors.

PATTEN, Philadelphia.

LES RECHERCHES EXPERIMENTALES SUR L'ENCEPHALITE AIGUE
EPIDEMIQUE (EXPERIMENTAL RESEARCH IN ACUTE EPIDEMIC
ENCEPHALITIS). P. HARVIER, *Rev. de méd.* **37**:353 (June) 1920.

The author reports his results in experimental inoculation of animals with epidemic encephalitis. The virus utilized came from the nerve centers of a woman of 45, who died from the disease on the ninth day. Fragments of gray substance, taken aseptically from the cortex, midbrain and medulla were triturated in normal saline.

The following conclusions are given: The virus of epidemic encephalitis is filtrable and is preservable in glycerine. Also, it maintains virulence after desiccation.

The virus is pathogenic for the rabbit and is inoculable into the rabbit by several routes, intracerebral, intra-ocular, by way of the peripheral nerves and by the nasal membrane, preferably scarified.

The virus is not pathogenic for the monkey or guinea-pig when it is obtained directly from man, but becomes so after several passages through rabbits. It differs, therefore, in this from the virus of poliomyelitis which is directly pathogenic to the monkey but not at all for the rabbit or guinea-pig.

There is no crossed immunity between poliomyelitis and encephalitis, and the serum of patients convalescent from encephalitis has no neutralizing action on the virus of poliomyelitis.

DAVIS, New York.

UEBER DAS VERHALTEN DER KNIEBENGER BEI DER ISCHIADI-
CUSLAHMUNG (THE BEHAVIOR OF THE HAMSTRINGS IN
SCIATIC PARALYSIS). D. GERHARDT, *Neurol. Centralbl.* **39**:3226 (May)
1920.

The author discusses vital resistance of the individual muscles when the motor nerve is damaged. The order and degree of susceptibility in the sciatic injury is the peroneal, anterior tibial and hamstring groups. He recorded a case of pachymeningitis lumbalis with loss of reflexes and paralysis of all the muscles of the legs, except the hamstrings and adductors.

Necropsy examination revealed an extradural inflammatory exudate in the region of the seventh and ninth dorsal vertebrae. The spinal cord showed a slight infiltration and degeneration of the root zone. The plantar reaction was absent; however, active contraction of the adductors and hamstrings was induced by plantar stimulation. Similar contractions were induced by passive movement of the legs backward and also by percussion of the quadriceps tendons, but the quadriceps failed to react. All muscles showed a prompt response to faradic stimulation.

This case substantiates the author's previous observation that by paralysis of the sciatic group, irritability of the peroneal and the tibial muscles may be lost, while that of the biceps group is retained. That the inflammation process was uniform over the lower part of the cord was indicated by the anatomic appearance, as well as the paralysis of the group above and below the biceps group. Although the biceps group is anatomically more closely associated to the peroneal and tibial group, yet physiologically it is in relation to the adductors, and its resistance to inflammatory attacks is also greater.

SHELDEN, Rochester, Minn.

UEBER ALTERNIERENDE. FAZIALISLAEHMUNGEN (RECURRING FACIAL PALSY). KURT BOAS, *Neurol. Centralbl.* **39**:567-571, 1920.

The author has assembled various studies showing that single recurrences in facial paralysis occur in from 3 to 7 per cent. Bernhardt collected sixty cases in which the patients had recurrences; 66.6 per cent. with a single recurrence, 31.6 per cent. with two recurrences, five with three recurrences and one with four, comprised the total.

He reports the case of a man, aged 30, who in 1906 developed right facial paralysis. A few days previous ten teeth were extracted from the right upper jaw. A few days later, the left facial nerve became paralyzed. The facial diplegia continued for about eight weeks, leaving slight bilateral ticlike twitchings. In 1913, following a short exposure during a car ride, he developed complete left seventh nerve paralysis, with recovery in four weeks.

Only so-called idiopathic cases are here considered. Several combinations of facial monoplegias and diplegia are recorded. Bernhardt deems a year interval as essential for a genuine recurrence. The shortest interval reported was twelve days, the longest eighteen years.

The author considers the bilateral ticlike twitchings as an evidence of over-irritability, viewing it as a predisposition toward paralysis. A favorable prognosis is the rule, this benignity being associated with favorable electrical reactions. Whether narrow fallopian canals or unusually vulnerable facial nerves are responsible, he is uncertain, but favors the latter theory.

SHELDEN, Rochester, Minn.

TONSILLECTOMY ON THE RECURRENCE OF ACUTE RHEUMATIC FEVER AND CHOREA: A STUDY OF NINETY-FOUR CHILDREN. W. ST. LAWRENCE, *J. A. M. A.* **75**:1035 (Oct. 16) 1920.

The author reports a study of 94 children, 85 of whom presented one or several of the manifestations of rheumatic conditions, and 58 of these suffered from organic heart disease. The cases were under observation from two to six years following tonsillectomy. The tonsillar conditions were variable. 13 per cent. markedly hypertrophied, 69 per cent. moderately so, and not enlarged

in 18 per cent.; recurrent inflammatory attacks were noted in 73 per cent., and recurrent "sore throat" in 7 per cent. after the removal of the tonsils; there were 20 per cent. of reoperations because of incomplete removal. The tonsillar lymph nodes were enlarged in 100 per cent. before operation and impalpable in 59 per cent. after operation. One or more attacks of rheumatic fever had occurred in 42 cases before tonsillectomy and there was no recurrence in 35 cases (84 per cent.) after the operation. Sixty-one cases showed myositis, bone or joint pains before the operation and no recurrence afterward in 47 cases (77 per cent.). Of the 58 patients with organic heart disease, 12 had suffered at least one attack of cardiac failure, with but one recurrence in 1 case after operation. Chorea was associated with rheumatic manifestations in 32 cases out of 40. In 3 cases chorea was the sole rheumatic manifestation, and attacks did not recur after operation, although on the whole the effect of tonsillectomy on the recurrence of chorea was not as definite as on other rheumatic manifestations. Forty cases showed recurring attacks of rheumatic fever before operation and no recurrence in twenty (50 per cent.) afterward.

PATTEN, Philadelphia.

NEURITIS OF THE CRANIAL NERVES IN LETHARGIC ENCEPHALITIS AND THE DIFFERENTIAL ANATOMIC DIAGNOSIS BETWEEN IT AND ACUTE POLIOMYELITIS. MONTROSE T. BULLOWS, *Arch. Int. Med.* **26**:477 (Oct.) 1920.

This is a complete clinical and pathologic report of three typical cases of lethargic encephalitis studied intensively as regards the pathology of the cranial nerves. The findings in the brain correspond to those described in all other cases in the literature, but in addition a special study is made of the cranial nerves. In the first case there was found a neuritis of both oculomotor and trochlear nerves and the right acoustic nerve; in the second case a neuritis of the optic, oculomotor, trochlear, trigeminal, abducens, facial and acoustic nerves, and in the third case a neuritis of the optic nerves, both oculomotor, left trochlear, both trigeminal and both acoustic nerves. Apparently the nerves involved bear no relation to the clinical symptoms. The author then gives his findings in more than fifty necropsies of acute anterior poliomyelitis in detail and sums up: "The difference pathologically, therefore, between lethargic encephalitis and poliomyelitis is evidently not to be found in the minute histology of the central nervous system but rather in the distribution of the lesions, the absence of marked edema and swelling of the spinal cord and the changes in other organs and tissues. . . . The neuritis of the cranial nerves, on the other hand, forms apparently a more distinctive difference."

WINKELMAN, Philadelphia.

EVOLUTION ET ASPECTS CLINIQUES DE LA DIPLEGIE FACIALE (EVOLUTION AND CLINICAL ASPECTS OF FACIAL DIPLEGIA).

A. DE CASTRO, *Rev. neurol.* **26**:80 (Nov.) 1919.

De Castro believes facial diplegia to be a much more frequent clinical occurrence than is commonly supposed, particularly the acquired peripheral type. He divides this type into two general classes, namely, facial diplegia observed in connection with polyneuritis and simple facial diplegia developing per se.

The first type is frequently found in association with polyneuritis of syphilitic origin and generally manifests itself after the extremities have become involved, affecting both sides of the face simultaneously or after an interval of several hours. The second type occurs almost entirely on a syphilitic basis (95 per cent.) and generally during the third or fourth month after infection. The paralysis develops on one side first, affecting the other in the course of several days, and is, as a rule, most severe on the side initially attacked. At any given time, it is rare to find both sides equally affected, nor is recovery usually symmetrical. The period required for recovery varies from a few days to several months. Postparalytic tic and contracture are relatively rare sequelae and, if present, are generally unilateral.

RAPHAEL, Kalamazoo, Mich.

LA PRESSION ARTERIELLE CHEZ LES EPILEPTIQUES (BLOOD PRESSURE IN EPILEPTICS). P. HARTENBERG, *Presse méd.* **28**:748 (Oct. 20) 1920.

Arterial pressures were taken with the Pachon instrument in eighty cases of idiopathic epilepsy. A few persons regularly showed hypotension or hypertension, but these departures from normal were not more frequent nor more marked than might be discovered in any other neuropathic group.

A number of attempts had been made by other writers to find some constant relationship between blood pressure variations and seizures of grand or petit mal. From this series no formula could be deduced. Whether a subject's usual pressure was high, low or average, it exhibited no behavior in the hours or minutes immediately antecedent, or subsequent, to an attack that could be construed as typical or pathognomonic. The pressure might rise or fall before an attack; it frequently fell afterward, but this was explicable by fatigue. Blood pressure studies of epilepsy apparently did not assist in clearing up questions of pathogenicity or of therapy.

HUDDLESON, New York.

NEUROPATHIC KERATITIS SETTING IN LONG AFTER ALCOHOLIC INJECTIONS INTO THE GASSERIAN GANGLION FOR RELIEF OF TRIGEMINAL NEURALGIA. CHANCE, *Arch. Ophth.* **49**:621.

The patient had had alcohol injected into the gasserian ganglion, April, May and June, 1916. In February, 1917, both the supra-orbital and infra-orbital branches of the trigeminal nerve were resected, and a few days later alcohol was again injected into the ganglion.

Almost three years later he developed edema of the cornea, with slight pericorneal and conjunctival injection, which condition the author attributed to the last injection of the ganglion with alcohol because it was not until the last injection that the patient was relieved of his tic.

From December, 1919, when first seen by the author, until May, 1920, the cornea of the left eye had become edematous, shed its epithelium and returned to normal three times. The iris was only slightly involved, and only a faint interstitial haziness remains at present.

The length of time intervening between the injection of the ganglion and the appearance of the keratitis was considered unusual by the author.

SCARLETT, Philadelphia.

EXPERIMENTAL OBSERVATIONS ON THE HISTOGENESIS OF THE SYMPATHETIC TRUNKS IN THE CHICK. ALBERT KUNTZ and O. V. BATSON, *J. Comp. Neurol.* **32**:335 (Dec. 15) 1920.

Various views are current regarding the source of the nerve cells which form the sympathetic ganglions. Some maintain that they are derived from peripheral mesenchyme, some that they migrate outward exclusively from the spinal and cerebral ganglions, and some that they arise from both the ganglions and the neural tube, in the latter case migrating outward along the ventral roots. The authors tested this last point by removing the neural crest and dorsal part of the neural tube in chick embryos before the sympathetic ganglions were formed. Sympathetic ganglions of approximately normal size are developed in the absence of spinal ganglions and dorsal nerve roots, provided the remnant of the neural tube is relatively large. The conclusion is that sympathetic neuroblasts do migrate out from the neural tube along the ventral roots, though migration in normal development from the spinal ganglions is not excluded.

HERRICK, Chicago

TRANSIENT HERPETIC RASH OVER THE AREA OF THE DISTRIBUTION OF THE OPHTHALMIC BRANCH OF THE FIFTH NERVE, FOLLOWING APPLICATION OF BLUESTONE IN A CASE OF PARINAUD'S CONJUNCTIVITIS. MATHIAS LANCKTON FOSTER, *Arch. Ophth.* **49**:621.

This article describes a typical case of Parinaud's conjunctivitis in a boy 14 years of age, with turgid mass of acuminate growths in the upper fornix. The area was treated with bluestone. Almost immediately a rash followed, resembling a fresh attack of herpes, over the left side of the forehead, sharply delimited by the median line. There was no pain or hypersensitiveness of the skin. The rash disappeared in two hours.

Another application of bluestone in three days was followed by a less marked eruption. Subsequent applications were followed by less severe eruptions until they ceased to appear.

SCARLETT, Philadelphia.

THE USE OF THE THERMOMETER IN MENTAL DISEASES. E. D. BOND, *Boston M. & S. J.* **183**:550 (Nov. 4) 1920.

In this day of refinements in diagnostic procedures, simple and established methods are apt to be neglected. Bond calls attention to the wealth of information which is to be gained from the habitual employment of the clinical thermometer in psychiatry. Of seventy-one consecutive hospital admissions, including all of the more common forms of mental disease, more than 50 per cent. showed a distinct febrile reaction. It is interesting to note that in manic-depressive psychoses the proportion of fevers reached almost 70 per cent. and in dementia praecox only 40 per cent. A group of seven "fever" patients making a transition from an undoubted infective state to a frank mania reveals a closer symptomatic relationship than is ordinarily expected and suggests a toxic agent as instrumental in the production of manic-depressive symptoms. In his recent annual address the president (Dr. S. DeW. Ludlum) of the Philadelphia Psychiatric Society remarked that on the first day of admission, patients in the psychopathic wards were so prominently stamped with the general signs of toxicity that it was practically impossible to venture a definite:

psychiatric opinion until the various eliminative channels had been set into motion. "Toxicity" like "shock" is a much abused term, but surely fever is to be regarded as presumptive evidence of its existence.

STRECKER, Philadelphia.

UEBER PUPILLENNYSTAGMUS (PUPILLARY NYSTAGMUS). LUDWIG and SCHILDER, *Neurol. Centralbl.* **39**:561-576, 1920.

The author deals with an exceedingly rare combination of rhythmical contractions of the pupils, synchronous and associated with nystagmus. In a case of encephalitis with bilateral external rectus paralysis, pupillary nystagmus, convergence nystagmus and twitching of orbicularis oculi were bilateral and synchronous. A woman, eight months pregnant, with eclampsia, awakened with right hemiplegia, dysarthria and a complicated combination of ocular paralyzes and nystagmus. With a vertical nystagmus in the left eye was associated a left pupillary nystagmus. In both cases the lesions were assumed in the tracts from Dieters' nucleus to the nuclei for the eye muscles.

Hippus is usually unattended by nystagmus although the association of both has been observed.

SHELDEN, Rochester, Minn.

CLINIC ON THE DIAGNOSIS AND TREATMENT OF NEUROSYPHILIS. H. C. SOLOMON, *Boston M. & S. J.* **183**:723 and 768 (Dec. 23 and 30) 1920.

The lessons to be drawn from Solomon's clinic on the diagnosis and treatment of neurosyphilis are that the central nervous system is involved comparatively frequently in both primary and secondary syphilis, but this involvement is ordinarily benign; a small proportion of meningitic and vascular syphilis occurs during the second stage; the proportion of neurosyphilis, tabes, paresis, meningitis, meningo-encephalitis, gumma and vessel disease may be placed at 10 per cent.; diagnosis is most uncertain without the aid of serology, and there is often a positive reaction in the spinal fluid long before definite symptoms are present; syphilitic meningitis, acute meningo-encephalitis and mild vascular conditions usually respond favorably to intensive intravenous therapy but in general the intraspinal method is superior.

STRECKER, Philadelphia.

PARALYSIS OF THE EYE MUSCLE AFTER ALCOHOLIC INJECTIONS FOR TRIGEMINAL NEURALGIA. J. FEJER, *Am. J. Ophth.* **4**:123 (Feb.) 1921.

The author cites two cases in which ocular paralyzes followed alcoholic injections, in the first case the abducent being affected and in the second, the oculomotor nerve. In the first case the injection was made in the region of the right malar bone and in the second in the region of the left supra-orbital notch. The author states that alcoholic injections are not so free from danger as Prof. Pichler maintains, and that many cases of neuromyolytic keratitis and atrophy of the nerves have been described in the literature.

REESE, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

The Three Hundred and Eighty-Fifth Regular Meeting, Jan. 4, 1921

WALTER TIMME, M.D., *President, in the Chair*

CASE OF LETHARGIC ENCEPHALITIS WITH UNUSUAL OCULAR SYMPTOMS AND NEARLY BILATERAL IMMOBILITY. DR. B. ONUF.

Dr. B. Onuf presented a case seen by him in his consulting service at Ellis Island, and diagnosed as lethargic encephalitis with resulting bilateral ophthalmoplegia and a slight titubation of gait, besides some muscular disturbances, of which later he expressed some doubt whether they were the result of the encephalitis or due to a juvenile muscular atrophy.

The data of anamnesis are somewhat meager: While coming from Italy to the United States, patient was taken with headache and what he called weakness of the lower extremities, being unable to stand and walk without support, and for three days he wanted to sleep day and night. He had also loss of appetite, but no fever. On Dec. 22, 1920, he was admitted to the Immigrant Hospital on Ellis Island, where Dr. Faulkner and Dr. Brown recognized the ophthalmoplegia and swaying gait and referred the case to the speaker for further neurologic examination and opinion. The patient on admission showed a slight elevation of temperature.

Neurologic Examination.—A neurologic examination was made on Dec. 24, 1920. The patient presented an ophthalmoplegia of both eyes. The eyeballs appeared to be quite immobile. Only when the patient looked into the mirror was there a slight convergent movement of both eyes, the right eye also moving slightly outward when following the mirror to the right side, while in the left eye only the convergent movement was noticed. There was a slight ptosis of the left side, but none on the right. Pupils showed slight inequality—the left was smaller than the right. Some light response was present in both eyes, but decidedly diminished in extent. Accommodative response was practically absent. Consensual reaction present in the right eye, but diminished, and was still more diminished in the left eye. There was no distinct asymmetry of facial innervation, but occasional twitchings on either side, more of the character of choreic tic than peripheral twitchings. The tongue was put out straight and showed no atrophy. The speech was of somewhat nasal character and was a little indistinct. The gait was swaying, with no preference to one side. There was a slight Romberg sign. The patient could not walk on a straight line on the floor, heel to toe. Knee, ankle and wrist reflexes were absent. The epigastric reflex was present on both sides and was fairly lively. The cremasteric reflex was slight on both sides. Plantar reflex caused flexion of the small toe and the one next to it on either side. The motor power of upper extremities was well preserved but there was a slight deviation in the index to index pointing test with eyes closed, also the index to nose

pointing test. There was slight adiadokokinesis. Touch, pain and localization were preserved everywhere. No areas of hyperalgesia were present. There was a slight degree of winged scapulae and lordosis which disappeared on sitting down. There was no pronounced atrophy of any group of muscles.

Mental Condition.—The patient appeared serious, perhaps slightly depressed, but prompt and to the point in all his answers to sensory tests. He gave promptly the day of the week, month and day of month. He said he was in a hospital, and that he came there two days ago (correct). He was intelligent and cooperated well.

Eye Grounds.—He had no optic neuritis, but the disks were perhaps slightly grayer on the temporal side.

Examination.—About 12 c.c. of cerebrospinal fluid were withdrawn by lumbar puncture. The fluid was clear and under normal pressure. This was made by Dr. R. P. Sandidge. It revealed: cell count, 4 cells per c.mm.; globulin, slightly positive; Wassermann reaction, negative. Dec. 30, 1920, the Wassermann test of the blood was reported negative. The urine was amber in color, acid, with a specific gravity of 1.025; no albumin; no sugar. Jan. 4, 1921: Blood count, by Dr. J. F. Paulonis, showed: red blood cells, 4,800,000; white blood cells, 7,800; polymorphonuclears, 71 per cent.; small mononuclears, 23 per cent.; large mononuclears, 4 per cent.; eosinophils, 2 per cent., and hemoglobin, 95 per cent.

In conclusion, Dr. Onuf expressed his gratitude to the medical staff of Ellis Island for the help given in working up this case and for the privilege of presenting it to the society.

DISCUSSION

DR. I. ABRAHAMSON said that at present he knew of no lethargic encephalitis cases, except chronic and remitting cases. Poliomyelitis and poli-encephalitis were in existence, presenting certain features of poli-encephalitis, but belonging to the group of poliomyelitis. Severe and complete ophthalmoplegias were unusual in lethargic encephalitis. The loss of knee jerks might be either of spinal origin or from lesion in Deiter's nucleus. The history of unsteady gait would indicate that the case might fit into the latter category. He had lately seen patients in whom there had been lethargy and diplopia, and finally total focal paralysis, whose cases he regarded as poli-encephalitis.

DR. WALTER TIMME said that there had been a recrudescence of lethargic encephalitis early in December. In the case presented, the number of cells was small for poliomyelitis.

DR. B. ONUF stated that he had thought of poliomyelitis, but the history of the lethargic condition decided him in favor of encephalitis. The patient had been taken ill on the boat coming from Italy, which might make a difference in the possibility of the condition being lethargic encephalitis.

DYSPITUITARISM WITH HYPERTENSION. DR. E. DAVID FRIEDMAN.

Dr. Friedman presented a student, aged 19, who for nine years had shown dwarfism and obesity, hypertension, and recently, recurrent pains in the region of the spine. The lad was the third of seven children. The parents and one sister were short and stout. One brother suffered from petit mal. The patient's history included measles and scarlet fever, but it was not known

whether the kidneys were involved at that time or not. The birth was normal and development normal up to the age of 9 years when he began to grow stout. There had been little gain in height since that time. The abdomen became pendulous, the fingers stubby, the feet remained small and the face became ruddy. Thyroid extract was administered in 1915 for over a year, and he grew 2 inches in height. The treatment was then stopped. The patient felt well and went to college. About six months ago he complained of shooting pains in the region of the spine and chest. Examination of the urine showed "kidney involvement." When presented he had shortness of breath and palpitation of the heart after exertion. Speech was somewhat hesitant and some impairment of memory had made itself apparent. Dimness of vision had been reported during the last two months and occasional headaches. There was nocturia but no polydipsia.

The physical examination revealed a short, obese man, 136.5 cm. in height (normal for his age, 175 cm.); the weight was 46.4 kilograms. There was erythema of the face and a pendulous distended abdomen. The teeth were somewhat spaced, the approximation poor, the lateral incisors projecting at a marked angle. The pupils and fields were normal. No sclerotic changes of the vessels of the fundus were reported. There were two pinpoint hemorrhages in the left fundus at one time. The thyroid was not enlarged. Examination of the lungs was negative; the heart was somewhat enlarged to the left and the aortic second sound was accentuated. The blood pressure was increased to systolic, 198; diastolic, 110. The genitals were small. There was an overgrowth of hair at the bridge of the nose, and the body was covered with fine lanugo hairs. The only pathologic neurologic findings were the slightly diminished deep reflexes, which may possibly be accounted for by the existent glycosuria and hyperglycemia. Other laboratory findings were normal. There was perforation in the septum of the nose, and the tonsils were moderately enlarged. The skin was dry and presented three distinct peculiarities: ringworm of the axilla and pubis, erythema and telangiectasis of the face, and striae distensae of the abdomen. Roentgen-ray examination showed undeveloped sinuses, the left being opaque. The lower molars were unerupted and impacted. Examination of the skull revealed a normal sella. The posterior clinoids, as well as the bones of the body and sphenoid bones were markedly atrophic and thin. A dense shadow was seen in the middle fossa, and a shadow at the root of the neck, extending up from the aorta, probably due to the large vessels. The roentgenograms of the hands showed bony development, such as is usually seen in a person of 13. Epiphyseal lines were still present. Libido was absent. The urine showed glycosuria. An electrocardiogram showed a left ventricular preponderance, and there were tachycardia and sinus arrhythmia. There was slowing of the rate on holding the breath and after vagus pressure. A slight alteration of the size and shape of the pulse wave revealed instability of the cardiac pacemaker. There was an unstable vagus and sympathetic balance. Intramuscular injection of atropin and pilocarpin produced no striking results. Epinephrin was not tried on account of the high blood pressure. The conjunctival epinephrin test of Loewi was negative. The title for the presentation was chosen to indicate the predominating features; but other glands were involved as well. The stunted growth points to a deficiency of the morphogenetic principle of the anterior lobe of the pituitary gland. The glycosuria and the hypertension might be interpreted as a hyperactivity of the posterior lobe. (The absence of a pathologic blood chemistry aside from the increased blood sugar and the presence

of a normal phthalein output would seem to exclude true interstitial nephritis.) The hypertrichosis and the increased cholesterolin content of the blood indicate some involvement of the suprarenals. The dry skin and delayed ossification of the bones show that the thyroid was not functioning normally. Reichmann¹ has designated a new pituitary syndrome differing from the two characteristic types hitherto described, hyperpituitarism producing acromegaly and hypopituitarism causing the dystrophy adiposogenitalis of Frölich. Reichmann's case resembles ours very much with the exception that his patient showed exophthalmos and bradycardia instead of the tachycardia seen in this case.

DISCUSSION

DR. WALTER TIMME asked whether the abdominal viscera had been examined. Dr. Friedman replied that with the exception of the palpation for the liver, the edge of which could just be felt, no examination had been made. Dr. Timme also asked whether the shadow of the chest had been considered from the point of view of a possible thymus shadow, to which Dr. Friedman answered that this point had been carefully considered and the possibility excluded. There was an absence of relative lymphocytosis characteristic of thymic cases. It was of interest, Dr. Timme also noted, that the genitals showed lack of differentiation.

DR. E. D. FRIEDMAN, in closing, said that the treatment had consisted of anterior lobe pituitary and thyroid extract for the past six months without any real change. The patient had received thyroid extract more or less constantly since 1915, the dosage varying from one-half to one grain three times a day.

JUVENILE TABES AND CASE REPORT TO ILLUSTRATE THE CONDITION. DR. CHARLES ROSENHECK.

Dr. Rosenheck sketched the development of the recognition of juvenile tabes as a distinct clinical entity from the day when Remak first reported three cases in 1885 and cited a discussion ten years earlier.

The difference between juvenile tabes and Friedreich's hereditary ataxia was not always apparent to earlier observers, and Hildebrandt first called attention to the error, drawing the distinction between such cases and those that might be considered true juvenile tabes. The diffidence of observers in accepting juvenile tabes as a true clinical entity is thought to have been due to the fact that up to 1910 no necropsy examination had been recorded to prove the existence of such a disease process.

The various factors that create the syndrome in the young and the respects in which it differs from the accepted orthodox type as observed in the adult are: The average of incidence is placed at 15 years. Mingazzini and Baschieri-Salvadori report an onset at 3 years of age as the youngest recorded case. Hereditary syphilis is responsible for the development of tabes in the young in the majority of cases. A few cases of infection in infancy are reported. According to Cantonnet's analysis, nearly twice as many females as males are affected, a striking reversal of conditions obtaining in later life. The mode of onset in a large number of cases was an early visual difficulty rapidly proceeding to optic atrophy and blindness. The percentage of this form of onset

1. Reichmann: *Deutsch. Arch. f. klin. med.* **130**: Nos. 3 and 4.

is much higher than in adults. Lancinating pains come next in order of frequency, 25 per cent. of the patients showing these as compared to 70 per cent. of adult patients. Bladder disturbances and gait defects were observed in fewer cases. Cantonnet remarks that ataxia in juvenile tabes is conspicuous by its absence, and the absence of marked gait disturbances in the tabetic blind has been commented on by all writers on the subject. Crises and girdle-like pains have not been observed at any time, nor have trophic phenomena been reported.

Thorough neurologic study renders the diagnosis a matter of simplicity. Cerebrospinal syphilis and Friedreich's ataxia are the only conditions that simulate it to any serious degree. Scoliosis, foot deformity, pyramidal signs, nystagmus, speech defects, etc., are never found in tabes, however; and the syndrome of cerebrospinal syphilis is somewhat different.

A young woman, 18 years of age, was next presented. She was the second of five living healthy children. Pupillary inequality was observed at the age of 12 by a school physician, and she was advised to wear glasses. The first subjective disturbances appeared at the age of 15 and took the form of severe lancinating pains. This was the only symptom for two years. Paresthesias, variable in character but always affecting the lower extremities, principally the toes, have been noted in the past year. There have been no visual difficulties during the period of the development of the malady. Examination of the patient revealed no abnormal attitudes of the voluntary motor system. The gait was somewhat disturbed by an ataxia which varied. In general, all coordinative activity was performed in a fairly creditable manner. Skilled test acts showed no gross defects. Deep reflexes were completely abolished; superficial reflexes were present and equal. No pathologic reflex could be elicited. The peripheral neural apparatus showed no pathologic alteration or impairment. Examination of the cranial nerves revealed that the pupils were fairly regular, markedly unequal and not responsive to light. Accommodative and consensual reactions were likewise abolished. The fundi showed a narrowing of the larger vessels and indistinctness of the smaller ones; the disk was grayish and showed clear evidence of early atrophy. Systemic examination revealed general deficiency in growth and development. Hutchinsonian teeth were present. Laboratory examination showed a positive blood Wassermann reaction. The spinal fluid contained 130 cells per c.mm.; globulin was increased; the Lange colloidal gold reaction was 5544334555, in addition to the positive Wassermann reaction. The prognosis in general, Dr. Rosenheck said, after summarizing the main conclusions reached, was excellent for life, but extremely poor for vision.

DISCUSSION

DR. D. J. KALISKI (by invitation) said that he wished to supplement the report by findings in an interesting case recently seen. There had been no diminution in vision until about the middle of last August, when the patient had suddenly become blind. Investigation showed that there had been symptoms soon after birth, but no treatment had been pursued after the child was 1 year old. There were many stigmas present, such as hutchinsonian teeth, complete optic atrophy, the Argyll Robertson pupil, which also reacted sluggishly, a positive Wassermann reaction and a positive cerebrospinal fluid. Cells were not greatly increased, but there was increased globulin, and a paretic curve resulted with gold solution. The child was becoming more and

more unruly, and the outcome will probably be taboparesis. A similar case was seen four or five years ago, in which the signs of tabes were clear. Taboparesis developed, and the patient died.

DR. ROSENHECK said that such cases were so rare, his being only the second that he had seen in twelve years, and the first certain one, that he regretted that fuller discussion had not been opened.

DR. ABRAHAMSON said that it would be interesting to study the endocrinous glands in the patient's family, to try to determine why one child was affected and the others spared.

DR. TIMME remarked that the girl showed numerous glandular disturbances, and the teeth especially were very small.

INCIDENCE OF ALCOHOLIC AND SYPHILITIC PSYCHOSES. DR.
GEORGE H. KIRBY.

Dr. Kirby drew attention to the fact that until recently one often encountered the statement that alcohol and syphilis together were responsible for from one quarter to one third of all cases of mental disease that required hospital treatment. During the past decade there have been certain developments which must exercise great influence on the prevalence and potency of these two important causes of mental disorder.

There can be little doubt that during recent years there has been a general movement against the intemperate use of alcohol. The federal prohibition amendment is but one indication of this movement. A striking reflection of this trend against inebriety is seen in the admission rate to the Bellevue Hospital alcoholic wards for the past eleven years. During this period the total number of cases of acute or chronic alcoholism (exclusive of the psychotic types) was 75,333. The high point was reached in 1910 when nearly 32 per cent. of all admissions to Bellevue Hospital were diagnosed as cases of alcoholism. Since then there has been a persistent decrease, the low point being reached in 1918 when a little over 6 per cent. of the cases were diagnosed as alcoholism. A slight upward turn of the curve in 1919 was due to a relative falling off of the general admissions to the hospital. The actual number of alcoholic cases in 1919 was less than in 1918. The figures for 1920 are not yet available, but preliminary estimates show a smaller number of cases than in 1919.

Dr. Kirby regarded the continuous decline in the ratio of alcoholic cases as all the more significant when one takes into consideration the increasing population of New York and the fact that there has been apparently no important shifting of alcoholic patients to other hospitals or institutions for treatment.

The percentage distribution of alcoholic psychoses admitted to all of the New York state hospitals also shows a decline for the past twelve years. A marked fall has occurred from the high point in 1908 (when nearly 11 per cent. of the state hospitals' admissions were diagnosed as alcoholic psychoses) to less than 2 per cent. in 1920. It was thought that the diminishing number of alcoholic psychoses reported might be accounted for in part by a change in diagnostic attitude; but the separate listing of the Korsakoff cases, which presents little room for diagnostic error, gave a curve which showed a decline similar to that for the other psychotic types.

Turning to the syphilitic psychoses, Dr. Kirby remarked that during the past ten or fifteen years a number of forces and influences have come into play which may possibly be lowering the incidence of syphilis as a disease or modi-

fying its later manifestations, especially the neuropsychotic forms. During recent years the public interest in syphilis, its prevention, consequences and treatment has been vastly increased. The therapy of syphilis has been revolutionized since the introduction of arsphenamin in 1910, and the lessened amount of alcoholism may perhaps also be reckoned as a favorable influence. Possibly it is too soon to expect to see results on account of the time which usually elapses after the infection before neurosyphilitic lesions become manifest.

Dr. Kirby showed a chart drawn up from state hospital data on cases of paresis admitted during the past twelve years. The ratio of these cases has held fairly uniform at about 13 per cent. of the total admissions. There has, however, been a slight fall during the past two years from the relative high point of 13.4 per cent. in 1918. Moreover, by using the recent census and calculating the rate of paresis per hundred thousand of the state's population, it appears that there has been a decline from 9 per hundred thousand in 1918 to 7.9 in 1920, the lowest figure on record. The actual number of cases of paresis admitted to the hospitals has also declined since 1918. This reduction may be somewhat significant in view of the steady increase in the population of New York State.

The data at hand would seem to justify the conclusion that alcoholism has declined perceptibly in recent years, and coincident with this decline there has been a marked fall in the number of alcoholic psychoses.

Mental disorder (paresis) due to syphilis reached a high point in 1918 with a subsequent decline in the relative and actual number of cases entering the hospitals.

DISCUSSION

DR. L. PIERCE CLARK opened the discussion of Dr. Kirby's paper with the suggestion that since dermatologists so frequently report that they see the early signs of syphilis at skin clinics, this would seem an excellent field for therapeutic work. If preventive workers would concern themselves with the earlier stages of the disease better prophylaxis would be obtained.

DR. B. ONUF asked how the diminution in syphilis and alcoholism has affected other psychoses. He thought it would be interesting to find out whether substituted drug habits, if such occurred, would also result in psychoses.

DR. L. P. CLARK asked whether the information collected by Dr. Kirby might not be turned over to mental hygiene publications so that the reading public might have correct data concerning the incidence of alcoholic psychoses and syphilitic psychoses and the relative decrease.

DR. B. ONUF said that Dr. Brill had spoken of a case of alcoholism treated by psychoanalysis with entire cure. The patient then proceeded to forge checks, so that Dr. Brill did not feel proud of his achievement and thought that the patient might have been better off as a drunkard. Dr. Onuf also said that Dr. Gregory had reported a recent great increase in cases of alcoholism admitted to Bellevue.

DR. IRVING J. SANDS said that his experience at Bellevue would lead him to believe that there was no such thing as a new form of psychosis resulting from prohibition. Furthermore, he stated that he had discussed that question with Dr. Barnes, and many of the older men who had charge of the alcoholic service in years gone by, and it was their impression that the old alcoholic repeaters were not now coming to Bellevue with a psychosis. It was true

that admission in general to the psychopathic ward was increasing, but that was due to other factors, and not to prohibition. He felt that prohibition had helped considerably.

Regarding the problem of general paralysis, he felt that the early recognition of neurosyphilis and the favorable impressions made by intensive treatment on mesodermic syphilis helped materially to lessen the number of parietic patients. He referred to Dr. Gregory's work on the modern conceptions of inebriety, which was published in 1917.

DR. D. J. KALISKI (by invitation) asked whether the decrease in parietic admissions to state hospitals had been brought about by the increase in facilities for treatment in other institutions. The state hospitals used to be the only refuge. Now more widespread and better known treatment centers and clinics, and the increase in treatment in private practice may have helped to lessen the admissions.

DR. KIRBY, in closing the discussion, said that he had seen no signs that other psychotic types were appearing as substitutes for alcoholic psychoses. The total admissions to the state hospitals have diminished, due, he thought, chiefly to the falling off of alcoholic psychoses. There has been no increase of drug psychoses; in fact, the number admitted to the state hospitals has decreased, although drug psychoses have always formed a relatively small group among the admissions to the state hospitals.

The suggestion that the decline in the number of syphilitic psychoses admitted might be due to more frequent treatment of these cases in general hospitals and in private practice, especially early cases of paresis, should be taken into consideration. Dr. Kirby did not feel, however, that this factor would modify greatly figures for the entire state over a period of years, unless actual curative results were being obtained in the treatment of early neurosyphilis. Unfortunately, experience shows that many cases of paresis that have been treated in general hospitals or in practice eventually reach state institutions.

REMARKS ON CONSCIOUSNESS IN THE EPILEPTIC FITS. DR. L. PIERCE CLARK.

The ordinary definition of the epileptic fit, Dr. Clark said, is a loss of consciousness with or without a convulsion. This accepted loss of consciousness, Dr. Clark finds, from patients' reports, to be questionable. One patient states: "When a petit mal occurs, I can usually control myself and go on talking or continue whatever I am doing. . . . There is a sort of turmoil in my mind. This would last only a second." Another says: "I feel intensely aware of myself." A third patient reports: "During the confused spell I have a feeling that I must not let any one know there is anything unusual, and that I must retain my poise at all costs. . . . Consciousness so far as outside affairs are concerned is suspended and my attention is directly turned in on myself." Another patient says: "I could always control these attacks if I really wanted to, but I let them come up. It was a gratification, I think, to something inside me."

From these statements, Dr. Clark infers that instead of there being a loss of consciousness in totality, the subject consciousness is increased. Awareness of surroundings may be greatly or entirely lost, but there is in its place heightened awareness of self. In more severe epileptic reactions the person may not be able to report his subject awareness; but from his appearance and manner it is apparent that he is in a state like that experienced by persons

able to record twilight states. Still more complete submergence of self to a condition resembling that of birth or earliest infancy is met with in deeper grades of disordered consciousness. The patient may answer questions and will always answer in disjointed sentences in the optative mood, such as "I am trying to," "I'd like to," etc.

The state of epileptic reaction, then, said Dr. Clark, is an involution of the normal development of consciousness. This latter is gradually evolved out of egoconsciousness to a rich combination of subject and object consciousness or to a unified composition of both. The epileptic reaction then has an enormous psychic importance, principally in the deteriorating influence on the power of sustained interest and attention, and the orderliness of normal objective life. Each attack refreshes and satisfies the patient's cruder egoconsciousness and enriches his egoistic interest. It is therefore to be expected that he will not be interested in recovering from his disorder and in foregoing his unconscious self gratification. To appeal to him, his personal satisfactions in everyday life must be increased beyond those obtained from his habit.

DISCUSSION

DR. JOHN T. MACCURDY thought that Dr. Clark had brought out an important clinical point in regard to the mental state of many epileptic patients. He agreed with Dr. Clark that particularly in cases with a prolonged aura a heightened sense of personal awareness was a frequent phenomenon. This is well illustrated in the account of an epileptic attack given by Dostoevsky in the "Idiot." On the other hand, Dr. MacCurdy wished to take exception sharply to Dr. Clark's contention that such heightened sense of awareness was in opposition to the loss of consciousness so generally recognized as pathognomonic of epilepsy. He insisted that consciousness was used in two senses. In the sense in which Dr. Clark was now using it, it meant a direction of attention to the patient's own ego. Clinically, however, consciousness usually means the reciprocal function of attention to the environment—objective consciousness, as Dr. Clark said. With any weakening of consciousness in the clinical sense there was always a tendency to an increase in the attention to the patient's own personality. In fact, this is the essence of introversion. A good example is seen in the normal phenomena of dreams. In them there is usually a heightened sense of the dreamer's personality, while objectively the subject is unconscious.

DR. GREGORY STRAGNELL (by invitation) said that in discussing the consciousness of the epileptic patient, the conscious, introverted attitude of the patient must be looked on as an escape from his obligations. It would hardly be analogous to unconsciousness. Two characters, famous in history, who were addicted to epilepsy, Napoleon and Dostoevsky, illustrate this. From the journals and letters of both of these, it is seen that both at about the age of 12 wrote home and threatened to simulate insanity to compel the state to look after them if they were not forthwith supplied with funds to maintain the position which they felt was their due. Each of these men found a compensation for an initial inferiority and the same form of retreat in later life when the carrying out of the compensation became too difficult. The mechanism in each of them was first the recognition of an initial inferiority of the ego which the one sought to compensate for by war, the other by literature; and if the

struggle became too difficult, they sought a retreat in the epileptic attack. This should give a clearer concept of the struggle going on in the consciousness of the person.

DR. C. P. OBERNDORF said that epilepsy was caused by many things and cured by many things. The term epilepsy does not mean very much. All stages of it may exist. In using the term, every form of convulsive attack is included. A case seen recently was that of a woman 42 years of age, the mother of ten children, who has attacks daily. In her case the epileptic attacks were due to an organ deficiency. Unless Dr. Clark can limit idiopathic epilepsy, due to a special type of physical disorders, his discussion will lead too far afield. To draw up a psychogenetic group will be undertaking something too misleading and will confuse the therapy.

DR. IRVING J. SANDS said that he considered Dr. Clark's contributions to the epileptic problem epoch making, and that all students of mental disorders were indebted to him. However, epilepsy, in his opinion, was an organic condition, and the fit was caused by the reaction of the brain to some stimulus. In his opinion there were definite structural (cerebral) alterations, macroscopic, microscopic or physiochemic in nature, caused by endogenous or exogenous toxins. He had had an opportunity to conduct an intensive study of epilepsy at the Manhattan State Hospital. The results obtained there by the administration of proper drugs convinced him all the more that epilepsy was an organic condition. In May, 1919, when sedatives, such as chloral, bromids, hyoscin and paraldehyd were used in controlling the psychotic epileptic patients, there were 502 recorded seizures; while in May, 1920, after the system of luminal therapy had been worked out, there were only eight seizures in the same ward. He stated that he had been able to follow up this therapy in Bellevue Hospital, where many of the epileptic patients had been sent because of some postepileptic unfavorable complications, and there too had obtained satisfactory results on that drug. There was no doubt but that psychogenic factors played an important part in many of the epileptic patients, especially in those having epileptic equivalents. Recently he had seen a case which might well fit into the group described by Dr. Clark. It was that of a 28 year old woman, a nurse, of unusual intelligence and ability, who was in charge of a big ward of mental cases. For the past four years she had been having attacks lasting one or two days, once every six months or so, which had been characterized by intense hemicrania, nausea, vomiting and scintillating scotomas. She was seen two weeks ago in one of these attacks. She left the ward suddenly, packed her trunks and cabled that she was coming home. She had had intense unilateral headache, nausea, vomiting and scintillating scotomas characteristic of migraine. After three days she felt greatly relieved. She stated that during this time she had thought only of herself, had had a sense of heightened self consciousness, and felt that several distressing conflicts that had been troubling her for a few weeks had been solved. She also added that all the other attacks would come on whenever confronted by some perplexing problem, and following these attacks she felt a sense of security in employing the mode of procedure indicated by her subconscious trend. This case Dr. Sands regarded as more or less of an epileptic equivalent associated with migraine.

DR. B. ONUF inquired concerning the relation between the injuries sustained during the attacks, and the theory of the wish of the person to escape from consciousness and to return to intra-uterine life.

DR. CLARK closed the discussion by stating that he emphasized time and again that he considered there was a definite group of essential epilepsy exclusive of groups involving organic conditions. He had always limited his remarks to cases in which no specific cause could be found. The epileptic fit essentially does not differ from conditions seen in the psychotic ward, and is a pure retreat to gain a certain result. Essential epilepsy is a mental disease with physical objective manifestations. Any diagnostic critique must keep this in view. It has been practically agreed that essential epilepsy may be considered a definite clinical phenomenon.

In replying to Dr. MacCurdy's comment, Dr. Clark said the sleep phenomenon was analogous but not parallel. As to medication, speaking from his own experience, he felt that luminal was probably the most insidious and destructive drug we have ever used. Its disastrous effects can be appreciated only after several years, whereas bromids reveal the harm they do on the surface after a short period. Luminal is deadly because of its character deterioration. It is worse than bromid medication since the mere stoppage of the drug does not do away with apparent deterioration, as in taking away bromid after prolonged use.

Epileptoid attacks, tabetic convulsions, etc., do not concern the idiopathic group. Their pathology may have been demonstrated, but until we obtain more data, every possible investigation should be continued.

COLORADO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 22, 1921

HOWELL T. PERSHING, M.D., *President, in the Chair*

Dr. Pershing thanked the Society for the honor of being chosen its first president; he predicted for the organization success and the accomplishment of much good for those interested in neurologic work.

A draft of the constitution and by-laws was presented and adopted.

ANXIETY NEUROSIS. DR. GEORGE E. NEUHAUS.

Dr. Neuhaus gave a brief review and history of anxiety neurosis as viewed originally by Freud and his successors, calling attention to the prominent characteristics which identified this clinical state. Four cases were reported, all of which were traced to sexual complexes, instancing recovery when the complexes could be brought out as the result of psychoanalysis.

DISCUSSION

DR. SAMUEL GOLDHAMMER referred to the cardiac type, comparing this to the neurasthenic, postinfluenzal and "gas" types, as observed in the army.

DR. EDWARD W. LAZELL said that the followers of Freud were making the same mistake as the older observers, and that while Freud was a clinician, that the phenomena he himself observed were not thoroughly interpreted. Quoting Kempf further that "the hormone is the messenger of the mind and not of the body," he expressed an opinion that all anxieties were regressions and the basis of the physical symptoms were the glands of internal secretions. Over-compensation of inferiorities is the chief cause of anxiety.

DR. C. S. BLUEMEL thought the type of individual was too often overlooked. Sexual complexes, he thought, were probably as frequent in other states if looked for.

DR. HOWELL T. PERSHING believes that these patients are subject to fear without a rational cause for the fear. All of us have the inherited mechanism which makes possible the development of fear. That these anxiety cases become especially sensitized depends on a neurotic inheritance, and some accident, shock or dynamic illness sets the matter off. Among the many other causes, the sexual may become operative, and in any such case the person may revert to what are regarded by him as errors, or indiscretions of youth in the same way as it may be developed in melancholia.

EPIDEMIC ENCEPHALITIS. DR. EDWARD DELEHANTY.

Dr. Delehanty spoke generally of the epidemic of encephalitis, referring to the wide discussion given the subject. In his opinion, it was a new disease; he had no recollection of seeing similar syndromes in previous years of practice. Several cases were related of rather atypical course.

DISCUSSION

DR. PHILIP WORK reported having seen six cases in which the pleocytosis ranged from twenty to sixty. In one of his cases a typical bulbar paralysis developed with a rapid course.

DR. HOWELL T. PERSHING referred to the previous epidemic of encephalitis in which he recalled cases which were regarded at that time as cases of meningitis; when the patients recovered the true nature of encephalitis was identified. He was impressed by the diversity of symptoms in the present epidemic. His first case resembled a Landry's type of paralysis until recovery began to appear in the reverse order—that is, the face recovered first. Another case was mistaken for melancholia; later a typical bulbar complication led to a correct identification. The third case appeared to be a poliomyelitis. Choreic and mental cases were also mentioned. The relationship of influenza is doubted.

DR. GEORGE A. MOLEEN called attention to a syndrome which he thought might be a form of noninflammatory hemorrhagic poli-encephalitis. He had found only one reference in the literature to this syndrome—this appeared in the *Revue Neurologique* under the caption "Syndrome of Tapia." Three cases were mentioned in all of which the left side was involved. A striking characteristic of all cases was the severe pain over the trigeminal area extending back to the occiput, which precedes the degenerative symptoms. This has been so severe as to have led to attempts at suicide in two instances. Shortly after the appearance of pain, atrophy was noticed in the same half of the tongue with marked furrowing and fibrillary twitching (of which photographs were shown); paralysis of the soft palate, the vocal bands and more or less weakness of the muscles of the neck was common in all cases. The paralysis of the ipsilateral vocal band was absolute. In one case only was strabismus noted. In one case there was a previous syphilitic basal meningitis from which the patient had recovered and negative Wassermann reactions of the blood and serum had obtained for a year; this was also true in the case reported from Brazil in the French literature. The other cases mentioned were free from syphilitic evidence. The treatment was similar to that for acute infec-

tions—symptomatic for the most part and with free alkalization and elimination. Complete recovery is the rule with the restitution of the tongue and restoration of the vocal power.

SAN FRANCISCO NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 4, 1921

MILTON B. LENNON, M.D., *President*

A CASE OF SUBACUTE MYELINIC DEGENERATION. DRs. E. W. TWITCHELL and R. W. HARVEY.

Drs. Twitchell and Harvey related the case of a man 52 years old with negative venereal history. In 1918 he had a severe attack of influenza. Four years ago he began to have tingling and a clammy feeling in his hands and feet. When examined on Aug. 15, 1920, he was so weak that he could not walk without a cane. Marked ataxia of the hands and feet was present. The tendon reflexes in the arms were diminished; those in the legs were absent. The lower abdominal reflexes were absent; the cremasteric reflexes were present but feeble. Blood count: red blood cells, 2,660,000; white blood cells, 5,800; hemoglobin, 82 per cent.; there were moderate poikilocytosis and anisocytosis. The blood Wassermann reaction and all spinal fluid tests were negative. A month later the patient was unable to stand or to feed himself. His hands ached so that he "wished they could be cut off." The entire body was very sensitive. In spite of this increasing weakness, the red count went up to 4,760,000 after a course of Fowler's solution and two doses of neo-arsphenamin. The patient died on Oct. 2, 1920.

DISCUSSION

DR. G. Y. RUSK reported that the brain showed a diffuse atrophy involving both frontal lobes and adjacent portions of the parietal and temporal lobes. The spinal meninges were normal, but on section the posterior columns showed a broad V-shaped brownish-yellow area of degeneration. Farther up the presence of chronic inflammatory changes suggested the possibility of syphilis in spite of the negative serology.

DR. E. S. DUBRAY, speaking from the internist's standpoint, said that he did not know of any particular group of cases that bring the internist, neurologist and psychiatrist so closely together as the group of which this case was an example. He discussed the spastic, ataxic and mixed forms and called attention to the fact that a review of all of the primary anemia cases at the Mayo Clinic had revealed the presence of neurologic signs in 80 per cent. The most valuable clinical sign that rules out syphilis is a stocking and glove anesthesia. He also cited a case of the kind under discussion which first had been thought to be one of multiple sclerosis.

DR. J. N. WOLFSOHN related a case of this disease in an alcoholic woman, seen by him in 1914, when he made a diagnosis of multiple neuritis. Three years later her symptoms persisted and blood examination revealed a red cell count of 800,000, with a color index of 1.4. After four months of arsenic therapy the red count rose to 4,000,000, but she had a flaccid paralysis with a positive Babinski sign and other features of beginning sclerosis of the cord.

Dr. W. F. SCHALLER said that he had frequently found cases of this disease. In 1917 he reported twenty-three cases with four necropsies in which the central nervous system was studied. None of his cases showed the unusual findings of this case, consisting of the perivascular and meningeal infiltrations rather than the cortical changes. His patients presented paresthesias rather than the pain complained of in this case. Many cases of this disease do not show anemia; frequently they show a high color index. Dr. Schaller said that when he has a patient with a spastic paraplegia and impaired sensibility, he watches the blood closely. He believes that if these patients are seen early enough something can be done for them. He tries arsenic and iron and colonic flushings, on the theory of intestinal intoxication. He said that he did not know of any disease that is so difficult to diagnose as combined sclerosis.

Book Reviews

PLASTIC SURGERY OF THE FACE BASED ON SELECTED CASES OF WAR INJURIES OF THE FACE, INCLUDING BURNS. With Original Illustrations. By H. D. GILLIES, C.B.E., F.R.C.S., Major R. A. M. C., Surgical Specialist to the Queen's Hospital, Sidcup. With Chapters on The Prosthetic Problems of Plastic Surgery by CAPT. W. KELSEY FRY, M. C., R. A. M. C., Senior Dental Surgeon, Queen's Hospital, Sidcup, and Remarks on Anesthesia by CAPT. R. WADE, R. A. M. C., Assistant Anesthetist, St. Bartholomew's Hospital. Cloth. Price, \$15. Pp. 408, with 844 illustrations. New York: Oxford University Press, 1920.

This admirable book deserves careful reading and study by every worker who is interested in plastic surgery. Major Gillies, as our readers know, took a leading part in bringing about the splendid results that were attained in the treatment of injuries of the face and jaws in England during the latter years of the war. It was largely due to his efforts and enthusiasm that patients requiring plastic repair of the face were gradually concentrated at Sidcup, where, with the assistance of his co-workers, the methods and success detailed in this volume were achieved.

Certain principles on which the author lays emphasis in his first chapter are so important that they will bear repetition, since they represent the essential considerations on which his work is based. They are: first, the necessity of making an accurate diagnosis at the very outset, by careful examination and palpation, by intra-oral and intranasal examination, by radiographic examination, and finally by an approximate reconstruction of the original on a plastic cast of the injured face; second, the importance of preserving normal tissue in its normal position until it is possible to attempt permanent repair; third, the avoidance of scar tissue formation, during the period immediately following injury, by suturing mucous membrane to skin about the border of large defects involving mucous cavities; fourth, the necessity of providing a lining membrane in reconstructing cavities which are normally lined with mucous membrane. Failure to observe the last principle, particularly in the reconstruction of the nose, will inevitably lead to failure, no matter how satisfactory the results may appear immediately following operation.

Concerning the implantation of foreign substances the author says, "The use of any foreign body is to be condemned whenever it is possible to substitute a graft from the patient himself." Metallic plates, celluloid plates, injection of celluloid, wax and paraffin have all been tried and found unsatisfactory. The use of bone grafts has gradually been restricted to the repair of defects of the mandible and malar bone. For cartilage the author has found an ever widening field of usefulness—in the reconstruction of the nose, in restoring the contour of the cheeks, of the forehead, etc. He says, "Cartilage, for large cosmetic purposes, stands unrivaled. It is available in sufficient quantity, is easily fashioned to the desired shape, and . . . remains permanently in the shape and size in which it is embedded."

The technic of covering raw surfaces, of providing skin flaps, and of the use of the Esser epithelial inlay is taken up in detail. The introduction of the latter, the author believes, marks a new epoch in plastic surgery and offers possibilities for wider application.

Separate chapters are devoted to repair of the cheek, injuries of the upper lip, injuries of the lower lip and chin, prosthetic appliances, injuries of the nose, injuries in the region of the eyes and ears, and the application to civil surgery of methods developed during the war. Of these chapters, that devoted to injuries of the nose deserves special attention. The author emphasizes particularly the basic principles that underlie successful work: provision of an external covering, a supporting structure, and a lining membrane. Only when all three are provided can failure be avoided. The technic of embedding the structural framework in flaps before transposing the flaps themselves is well illustrated.

A wide variety of illustrative cases has been selected in the discussion of the treatment of different types of injuries, and the technic of operation has been detailed with diagrams and admirable illustrations. The types of injury described are so varied that the surgeon who is confronted with specific problems will find valuable suggestions and assistance for his own work as he reads these pages.

This volume stands as a mute but powerful argument for a league to end war. As long, however, as wars continue, the art of surgery and the wounded will owe much to the industry and imagination of men such as the author and his colleagues.

SLEEP WALKING AND MOON WALKING. A MEDICO-LITERARY STUDY. J. SADGER, M.D., Vienna. Trans. by Louise Brink. *Nervous and Mental Disease Monograph Series*, No. 31. Pp. 138. New York.

In this monograph Dr. Sadger has attempted to discover the cause and meaning of these phenomena of sleep: the motor responses to dreams. He uses as sources for his conclusions one exhaustively analyzed case, one case in which the sleep walking element was a minor one, three cases quite briefly described, and three autobiographic accounts. This, the clinical part of the book, occupies the first forty-four pages; part 2, almost 100 pages, with the exception of a couple of pages of résumé and conclusions, is taken up with an analysis of episodes of sleep walking found in the literature of fiction and drama. In the clinical part there are long quotations from conversations between the patient and the doctor tending to show the causal relation between infantile sexual memories and sleep walking. There is also evidence adduced showing the relation of the moon to the light borne by the parent visiting the baby's crib. In the first case analyzed the patient was able to remember pretending to be asleep and walking to the parents' bed, a thing she later did in her sleep. She pretended to be asleep so that she would not be held responsible. The sleep walking served the same purpose. The analysis of sleep walking shows how unconscious, one might also say instinctive, is the necessity of mankind to escape a sense of responsibility. It is this necessity which creates much of the conflict resident in the sexual problem. In the tales analyzed in the second part of the book the same principles are found; a gratification of impulses for which the individual would escape responsibility, a uniformly sexual character of these impulses and a tendency for them to refer back to childhood. The erotic character of infantile urination is touched on in the first case analyzed and indicates the line, according to the author, along which investigation of the problem of nocturnal enuresis of the adolescent is to be followed. Acceptance of Freud's theory of dreams is a prerequisite to a sympathetic reading of Sadger's exposition, and there is much in the book that only psychanalytic devotees will accept. To believe that an

infant of 1 year is sexually stimulated by mere contact with the mother and that this is remembered in adult life, would be difficult for one not a Freudian disciple. To base the assertion of "expulsion of a secretion from the cervix uteri" during supposititious sexual erethism in a child of 2 years, on a statement of the patient grown to be a neurotic woman, does not appeal to the ordinary neurologist.

The introduction and analysis of works of fiction to aid in proving a scientific hypothesis doubtless will surprise and fail to appeal to the ordinary medical reader; to a follower of the psychoanalytic school the value of this sort of work is conceded. The inherent difficulty of getting any person's mind to follow associations that are bound to lead to embarrassing or distressing self-revelations is avoided by an author who follows such associations in his own mind, but tags them with the name of one of his characters. Dr. Sadger's work deserves the careful reading of any one interested in an attempt to deliver the mental side of medicine from the realm of the mystical according to the conceptions and methods of the Freudian school.

The translation is generally clear but the English is often quite Teutonic in construction.

DIAGNOSE DER SIMULATION NERVOESER SYMPTOME AUF GRUND EINER DIFFERENTIALDIAGNOSTISCHEN BEARBEITUNG DER EINZELNEN PHAENOMENE. Ein Lehrbuch für den Praktiker von PROF. SIEGMUND ERBEN, M.D., Wien. Mit. 25 Textabbildungen und 3 Tafeln. Zweite, vielfach ergänzte und erweiterte Auflage. Pp. 254. Berlin: Urban & Schwarzenberg, N. Friedrichstrasse 105 b. Wien., I. Maximilianstrasse 4, 1920.

The first edition of this monograph appeared in 1912 and was based on a course of lectures to medical officials and physicians of workmen's beneficiary societies (Kassenärzte), the author stating that "for the majority of practitioners the detection of simulation has become very important." So far, in this country, the detection of simulation has not been a very important function of the general practitioner but with the generalization of workmen's compensation laws, it remains to be seen whether or not simulation will become an everyday affair.

Naturally the author's task falls into three parts: means of detecting simulation of (1) organic disease, (2) functional disease and (3) psychoses.

This task Professor Erben has performed very well. In the preface and first chapter he intimates that he will clearly show how to distinguish simulation from functional nervous disorder, and this he does about as well as any one can—which is to say not well at all.

As indicated by the title, the subject is approached through the signs and symptoms belonging to each disease or disorder considered, and then these are contrasted with the findings in simulation. What is perhaps equally important, the author shows how simulation frequently is correctly assumed.

The first chapter is on nervousness and neurasthenia and is principally an exposition of the symptoms shown by the neurotic. Considerable stress is laid on the author's "vagus sign" as indicative of the hypersensitiveness of nervous people. With the head strongly inclined forward, the patient squats (niederhocken) when the pulse quickly slows; on rising the rate slowly increases to above the normal. This is repeated five or six times. The result is more striking if the pulse rate has first been raised by exercise. This sign is said to be valuable in distinguishing functional from myocardial tachycardia. Many

other excellent tests are given for deciding between organic disease and functional disorder but very little to help one to separate simulation from hysteria.

The chapter on pain and hyperesthesia is excellent, containing many helpful hints on a subject always difficult and in medicolegal cases often most perplexing. Here, too, the author notes some organic things (Bernhardt's "laborer's pain, flat foot") apt to be overlooked. For the detection of simulated tenderness, the tests of Müller, Mannkopf-Runpf, Parrot and Selligmüller are given, with the opinion that if they are positive there is tenderness, but that if they are negative, tenderness cannot be excluded.

There is a good discussion of backache, sciatica and other leg pains and anomalies of carriage and gait. Coming from the home of Bárány, one would expect the book to cover vertigo and dizziness. It does, in twenty-two pages.

The trained neurologist and the experienced industrial doctor will find in this work little that is new, but these specialists are not numerous. Other practitioners will find much with which they are not familiar and much to help them out when the occasional medicolegal case or returned soldier claiming recompense turns up.

Evidently the work is based on extensive experience and earnest work, but aside from a number of citations from Charcot, the valuable contributions of those outside of Austria and Germany are conspicuous by their absence.

A TEXTBOOK OF PHARMACOLOGY AND MEDICAL TREATMENT FOR NURSES. By J. M. FORTESCUE-BRICKDALE, M.A., M.D. (OXON.), M.R.C.P. (LOND.); Captain, R. A. M. C. (T. F.); Physician to the Bristol Royal Infirmary and Clinical Lecturer in the University of Bristol; Formerly Lecturer on Pharmacology in the University of Oxford. Pp. 371. London, Henry Frowde, Oxford University Press; Hodder and Stoughton. 20 Warwick Square, E. C. 4.

This book was written for the purpose of explaining to nurses the principles of medical treatment and is divided into two parts. In the first section, devoted to pharmacology, an attempt is made to convey in "simple terms" the known facts regarding the action of various drugs on the animal body. The second section deals with the treatment of disease.

While there is much that is commendable in the work, there is, unfortunately, much to be criticized. To discuss chlorids, bromids and iodids under the heading of chlorin, bromin and iodin, is not in keeping with up-to-date pharmacologic teaching. Again and again the author describes medicinal products that are not in general use or that are of no therapeutic value. This is entirely out of place in a book intended for nurses, for whom an acquaintance with the really useful drugs is all that should be required. On the other hand, nothing is said about dosage of drugs, a subject that nurses surely ought to know something about.

Likewise, in the section devoted to treatment, the author's tendency to encyclopedic diction is to the fore. But one misses explanations as to the nature of the diseases discussed, which is, of course, not really required in a book purely devoted to therapeutics; but what need has a nurse for a study of pure therapeutics?

To present the facts of a science simply, interestingly, and in a manner suitable for those who require or desire merely a general introduction to a subject, is an art mastered by comparatively few. Fortescue-Brickdale is not one of these.

COMMON INFECTIONS OF THE KIDNEYS WITH THE COLON BACILLUS AND ALLIED BACTERIA, BASED ON A COURSE OF LECTURES DELIVERED AT THE LONDON HOSPITAL by FRANK KIDD, M.B., B.C. (CANTAB.), F.R.C.S. (ENG.), Surgeon to London Hospital; Surgeon-in-Charge of Genito-Urinary Department, London Hospital; Member of the International Society of Urology; Membre de l'Association Française d'Urologie. With an Additional Lecture on the Bacteriology of the Urine by Dr. Philip Panton, Clinical Pathologist, London Hospital. Pp. 314. London: Henry Frowde, Oxford University Press; Hodder and Stoughton, 20 Warwick Square, E. C. 4, 1920.

This book is based on a series of lectures delivered by the author and deals with the common infections of the kidney, as well as infections of the prostate and testicle, about one half being devoted to the kidney. It is written in the author's usual clear, concise manner and gives the present-day views regarding the various lesions under discussion. The theoretical considerations are included in one chapter, an arrangement of distinct value in this type of book which, as the author says, is to serve the busy practitioner. The histories of cases on which the book is based are given in an appendix.

In his consideration of the treatment of the various types of colon infections of the kidney the author differentiates between those calling for surgical treatment, which he states comprise a small group, and those suitable for nonoperative management. The author is very much in favor of treating cases of colon bacillus pyelitis by means of pelvic lavage with silver salts. Much emphasis is laid on accessory treatment in these infections, such as proper attention to the gastro-intestinal tract, regulation of the diet, warm underclothing, etc. He does not believe that vaccines are of value, laying great stress on the administration of liberal doses of alkalies.

The chapters dealing with blood-borne infections of the prostate and testicle are well written, and attention is called to the possibility of infections in these organs being due to organisms other than the gonococcus. Failure to recognize this fact results many times in patients being falsely accused of having gonococcus infections.

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PSYCHIATRIC ASPECTS OF EPIDEMIC ENCEPHALITIS*

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AND

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Since the earliest descriptions of epidemic encephalitis, most observers have recognized that some degree of psychic disturbance formed an essential feature of the clinical picture. Very few studies have, however, dealt particularly with the psychiatric aspects of this disease. In fact, a review of the extensive literature which has appeared during the past few years shows, in contrast to the intensive study and finer differentiation of the various neurologic syndromes, that the psychotic manifestations of encephalitis have on the whole received relatively little attention.

Those writers who have reported psychiatric observations have been concerned mainly with the initial or early mental symptoms, and remarkably little has so far been recorded as to the course and outcome of the mental disturbance; and the important questions of permanent mental damage and the more chronic psychotic manifestations have been discussed hardly at all. It is, of course, evident that a satisfactory solution of certain of the psychiatric problems presented by the disease will require a longer period for clinical observation of cases than has elapsed since the appearance of the epidemic in 1918-1919.

The material on which the present communication is based has been selected to meet, so far as possible, the requirements of a well-rounded psychiatric study. The group available for the analysis comprises eighteen cases. We have included only those about which there could be no doubt as to the diagnosis of lethargic encephalitis. Moreover, they are all cases about which we were able to get a fair anamnesis from relatives or friends. We were fortunate in being able to study cases in both general hospitals and state hospitals for the insane. We

* An abstract of this paper was read before the meeting of the Association for Research in Nervous and Mental Diseases, New York City, Dec. 28 and 29, 1920.

were thus enabled to compare early and late clinical pictures, and by looking up the patients discharged from the hospitals, we secured additional data on which to form an opinion as to the course and outcome of the disorder in each of the patients included in our series.

FREQUENCY OF SEVERE OR PROLONGED PSYCHOSES

It is probably true, as suggested by Barker,¹ that during the present epidemic many cases of encephalitis have been overlooked and undoubtedly many cases of psychoses are passing through the hospitals without their relation to encephalitis being suspected. We know of several cases of acute psychosis which were not recognized as an encephalitic disorder until the brain was studied postmortem.

In New York State most of the cases in adults with prolonged or severe psychoses eventually reach the state hospitals. The staff physicians of the institution have been, since the epidemic was recognized, on the alert for cases of the disease.² It is, therefore, of interest to learn how many cases have been received in the state hospitals. So far the number admitted because of grave psychoses is apparently small in comparison to the number of cases reported in the community and treated in general hospitals. During the hospital year from July 1, 1919, to June 30, 1920, in only twenty cases was the diagnosis of epidemic encephalitis considered probable among the 6,500 admissions to the New York state hospitals.³

CLINICAL GROUPINGS

The wide variety of terms used by different writers to designate the more striking psychotic manifestations of encephalitis indicates a lack of any well founded nosologic conception. The following list, by no means an exhaustive one, taken from recent articles, illustrates very well the wide range of designations applied to the mental disturbances accompanying or following epidemic encephalitis: acute delirium, maniacal delirium, afebrile mental confusion, amentia, mania, true mania, manic state, real hypomania, anxiety depression, excitable

1. Barker, Cross and Irwin: Epidemic Acute and Subacute Nonsuppurative Inflammations of the Nervous System, *Am. J. M. Sc.* **159**:157, 1920.

2. New York State Hospital Quarterly, November, 1920, contains an article on "Encephalitis Lethargica" by Drs. Montgomery and Waldo, of the Willard State Hospital and a report of five cases by Dr. Heyman of the Manhattan State Hospital.

3. Unfortunately, we have no data as to the number of encephalitis cases which developed in New York State during this period. We are informed by Dr. E. S. Godfrey of the state board of health that epidemic encephalitis was not made a reportable disease until June 1, 1920. During that month, the epidemic being already on the wane, only sixteen cases were reported in the state.

depression, melancholia, emotional stupor, hebephrenia, catatonia, lucid catatonic stupor, paranoid state, grave delusional psychosis, fully elaborated psychosis, epileptico-maniacal psychosis, Korsakoff's disease, hysterical attack and psychoneurosis.

It is unfortunate that psychiatric terms already identified with well-established clinical entities should have been so freely used even for descriptive purposes. Some observers, however, apparently do not use the terms merely in a descriptive way, as they seem to think that manic-depressive insanity, dementia praecox, paranoiac states and other constitutional psychoses may be definitely related etiologically to epidemic encephalitis.

Only a few observers have so far offered any systematic psychiatric grouping of the cases studied. Indeed, many writers have been content merely to mention and keep apart as "psychotic forms" or "mental forms" those cases in which the psychic manifestation overshadowed the neurologic syndrome, although admitting that in any of the various types, separated on anatomic grounds, a mental disturbance may exist. Some authors, as Buzzard, ignore the mental aspect of the disorder and adhere to a strictly anatomic classification. Others, as Tilney and Howe,⁴ group their cases partly from the anatomic and partly from the psychiatric point of view. Hesnard⁵ has perhaps attempted the most comprehensive grouping of psychotic syndromes. His main divisions are:

I. Mental disorders in the more common neurologic types. The mental disturbance, assuming a subordinate rôle, usually does not go beyond a state of lethargy or mild confusion.

II. Mental disorders in a group of cases in which the neurologic signs are less prominent while the psychotic disturbance stands in the foreground from the first. These are the "encephalitic psychopathies" for which the general designation of "acute psycho-encephalitis" is suggested.

As clinical varieties under the second division the author mentions the following:

1. Psychosomnolent form: Drowsiness succeeded by mild excitement (hypomania) passing into confusion which is later followed by depression.

2. Lucid catatonic stupor: General immobility, catatonia, preservation of mental clearness.

4. Tilney, F., and Howe, H. S.: *Epidemic Encephalitis*, New York, Hoeber, 1920.

5. Hesnard: *La Psycho-Encéphalite Aiguë Epidémique*, *l'Encéphale* **15**:443 (July) 1920.

3. Acute delirious form: The delirium may be frankly hallucinatory, analogous to febrile delirium tremens.

4. Confusional form: Marked reduction of mental activity with clouding of the sensorium.

5. Korsakoff form: Disorientation with confabulation, often, but not always, accompanied by neuritic or poliomyelitic symptoms.

6. Miscellaneous psychic disturbances: Especially sequelae seen most often in children who show mental dulness or various changes in character and disposition.

An obvious objection to any such clinical grouping as the foregoing is that it tends to set up a false contrast between neurologic and psychotic syndromes. It arbitrarily leaves largely out of consideration the mental disturbances, often severe, which frequently accompany the more marked neurologic syndromes and tends to give the impression that when mental symptoms are pronounced the physical symptoms are likely to be slight and vice versa. We do not think there is any justification to speak of "psycho-encephalitis" and thereby suggest that the mental reaction in certain cases is more important than the physical aspects of the disease. A patient with mild mental symptoms at the beginning may later on show a marked psychotic disturbance and it is well known that the initial physical symptoms form no criteria of what the eventual neurologic syndrome may be.

Later we will show that the so-called "psychosomnolent form" and "lucid catatonic form" are not clinically distinct types. They are only in a very superficial way different from the other types, may occur during the course of any of the forms mentioned, all of which we find contain elements fundamentally more important in psychiatric differentiations than those given by Hesnard.

PSYCHIATRIC PROBLEMS OF EPIDEMIC ENCEPHALITIS

It is now known that epidemic encephalitis is an acute or subacute inflammatory disease of the central nervous system, a meningomyelo-encephalitis. It is probable that the disease is caused by an infectious organism. Furthermore, it seems reasonably certain that as a result of the activity of the infective agent or its toxin within the body a varying degree of temporary or permanent damage to the nervous tissues takes place. As a consequence, psychic disturbances of varying degrees of intensity arise.

The psychiatric problems of epidemic encephalitis would, therefore, appear to lead directly to a consideration of those types of mental disturbance susceptible of being called forth by toxic-infectious causes which act deleteriously on the central nervous system. From the standpoint of clinical differentiation the first question might be: What are

the known characteristics of mental disorders arising on such an etiologic basis?

The studies of Meyer,⁶ Bonhoeffer,⁷ Hoch⁸ and Bleuler⁹ have shown that disturbance of mental function, like disturbances of other functional mechanisms, takes place in a relatively limited number of ways. Clinical studies have in fact made it possible to circumscribe a fairly small number of abnormal mental reaction types now recognized to be of fundamental importance in all questions of pathogenesis and symptomatology of mental disorders. So far as we can see at present, there are three mental reaction types which are of special psychiatric significance. These are:

1. *Organic Reactions*.—These are characterized essentially by impairment of apprehension, interference with elaboration of impressions, defects in orientation and retention; difficulty in activation of memories with variability in mental capacity and level of attention—the so-called mental tension defect. It is now well established that this reaction is seen only when the brain has been damaged, temporarily or permanently, including injury by toxic-infectious agents.

2. *Affective Reactions*.—These are characterized by emotional disturbances which follow essentially the pattern of the normal affective states. Typical examples are the manic-depressive oscillations and other benign emotional disorders.

3. *Trend Reactions*.—These are characterized by abnormal trends and ideas, an unusual attitude toward the outside world, with constitutional peculiarities and psychogenic mechanisms in the foreground. Examples are schizophrenic and paranoiac states.

While these mental reaction types are often encountered clinically in pure form, they need not coincide with what we call "mental diseases." In fact, one reaction does not necessarily exclude another. For instance, an affective or trend reaction may be superimposed on, or released by, an organic reaction, so that the resultant clinical picture is thereby given a special cast. Descriptive psychiatry has been, to a considerable extent, concerned with an effort to single out and identify as clinical entities groups of symptoms which belong to one or the other of these forms of mental reaction.

6. Meyer: The Problem of Mental Reaction Types, Mental Causes and Diseases, Psychological Bull. 5:245, 1908.

7. Bonhoeffer: Die Symptomatischen Psychosen in Gefolge Von Akuten Infektionen und Inneren Erkrankungen, Franz Deuticke, 1910.

8. Hoch: The Problem of Toxic-Infectious Psychoses, Bull. New York State Hospitals, November, 1912.

9. Bleuler: Dementia Praecox Oder Gruppe der Schizophrenien, Franz Deuticke, 1911.

It is our view, based on pathology and probable etiology, and also on clinical experience, that the psychiatric problem of epidemic encephalitis belongs essentially in the realm of the organic, more specifically the toxic-infectious mental reaction types. The present study is a clinical contribution in affirmation of this conception. Before proceeding to discuss our case material, we will sketch briefly the principal features of the organic mental syndromes.

The conception of an organic syndrome is not a new one in psychiatry, although the old and unsatisfactory designation of "organic psychoses" passed out of use as we became better able to differentiate various types of brain disease. A more precise description and designation of the organic reaction has resulted from the recent work of Kraepelin¹⁰ and of Bonhoeffer⁷ who have analyzed in detail the features common to all psychoses which develop in reaction to brain disease or injury. Bonhoeffer especially has extended our knowledge by studies of "exogenous" causes and the clinical pictures constituting the symptomatic psychosis, that is, those due to bodily diseases and poisons.

The *chronic* organic reactions are seen in arteriosclerosis, senile deterioration, chronic Korsakoff disease, paresis, brain tumor, etc., all of which show interference with mental grasp, diffuse memory defects, impairment of retention, variability in capacity and clearness, diminished ability for complicated thinking, and fluctuations in levels of attention—a typical mental tension defect.

The *acute* organic reactions have recently become better understood, chiefly as a result of Bonhoeffer's work on acute Korsakoff's disease, delirium tremens and other deliria accompanying physical diseases. The typical acute reactions are the deliria due to trauma, alcohol, uremia, lead and carbon monoxid poisoning, infections, bacterial toxins, etc. Brain torpor, somnolence or stupor may, however, be the most striking clinical expression of the reaction, replacing or alternating with the delirium. In the acute types, we encounter the difficulty in mental grasp and elaboration of impressions to the point of clouding of the sensorium, orientation and retention defects, and a striking variability in the level of consciousness: when lowered the train of thought becomes fragmentary (muttering and incoherence), when raised by questions and external stimuli the patient becomes more attentive. With clouding of consciousness, dreamlike ideas and hallucinations appear, and there is a marked tendency for habitual trends of thought and activities to determine the scene—the well-known occupation delirium.

10. Kraepelin: Psychiatrie, Ed. 8, 1910.

Psychogenic deliria in contrast to organic deliria, as pointed out by Bleuler⁹ and by Hoch,⁸ present quite a different type of reaction. In them the reaction centers about the realization of a wish with more or less complete splitting off of the rest of the personality, excluding, so to speak, the outside world or falsification of reality in harmony with the main trend.

As already intimated, even in the purest organic reactions, it is necessary to consider various modifying elements, particularly the personality make-up, the special etiologic factor at work, the intensity and duration of its action and predilection for definite parts of the brain. The widespread cropping out of constitutional traits and the appearance of various affective and trend reactions in the organic psychoses need not be dwelt on at present.

In concluding this part of the discussion we will simply mention the more common clinical forms of the acute organic reactions as they occur in psychiatric practice. Later we will discuss these types in relation to the symptom-pictures encountered in encephalitis.

1. Acute delirium: the organic features of which are plainly evident.
2. States of psychic torpor: various grades of mental dulness, somnolence, stupor or coma.
3. Amnestic-confabulatory complex: acute Korsakoff syndrome.
4. A group of more complicated psychotic reactions to which the term *amentia*¹¹ is often applied. In these the organic elements, disorientation and clouding of the sensorium, are combined with emotional disorders, trends and psychomotor disturbances which are not usually so prominent in the other acute organic syndromes. In these more complex cases, the personality traits and various psychogenic reactions come more to the front and thereby give a special cast to the clinical picture.

GENERAL SURVEY OF CASE MATERIAL

In Table 1 are presented the principal features of the neurologic and psychotic syndromes as encountered in our eighteen cases.

By far the most constant physical symptom in this series of cases was disturbance in the oculomotor mechanisms alone or in combination with other physical signs. This was plainly evident in all cases except one which was a pseudoparkinsonian type (Case 6).

Next in frequency among physical manifestations were pseudoparkinsonian symptoms, noted in six patients (Cases 3, 4, 5, 6, 7 and 11).

11. The term "*amentia*" is not free from objection, but it is a useful designation in the sense here indicated for a clinical group seen in psychiatric hospital practice.

In two cases, there were symptoms of a transitory nature pointing to the cerebellum (Cases 9 and 11).

In two patients, a thalamic type of facial weakness was noted and in one of these there was uncontrollable laughter (Cases 5 and 12).

In one patient, meningeal symptoms were prominent at the beginning (Case 15).

In one patient, a lasting hemiplegia developed during a prolonged stupor (Case 17).

In eight cases, the physical symptoms have cleared up entirely, leaving ten cases in which there are light or severe physical residuals.

In general, we were not able to make any correlation between types of physical symptoms and the form or outcome of the mental disturbance. It is, however, interesting to note that of the ten patients with physical residuals not one can be said to have recovered completely mentally. On the other hand, of the eight patients without physical residuals, two have fully recovered mentally.

A general survey of the psychotic syndromes reveals that in every case there was at some time during the clinical course a period of pathologic drowsiness or mental torpor varying in degree from abnormal sleepiness to stupor or coma. The fact that this symptom appeared in all of our patients tends to make us think that cases in which it does not occur at some time during the course of the disease are the exception. Its appearance is very irregular. It may constitute the chief feature of the mental picture or it may appear for a brief period only during the course of other mental disturbances. Thus, we find it as an initial symptom, or following a delirium, excitement or period of insomnia, or it may immediately precede the convalescence.

The next most striking feature of the psychotic picture was the appearance of delirium or mental confusion. This symptom occurred with varying intensity and duration in fifteen of our eighteen cases. In the three cases in which it was not apparent, we do not, however, feel that delirious features in the sense of a temporary alteration in the level of consciousness with impairment of mental grasp, etc., can be definitely excluded. These three patients, unfortunately, could not be sufficiently examined for the lighter grades of impairment of the sensorium and delirious "dips" (Cases 1, 4 and 5). No patients presenting an acute or chronic Korsakoff mental picture have been observed by us.

A point of special interest is the appearance of trend reactions and emotional disturbances outside a setting of delirium or mental confusion. In general, the persistence of a delusional trend after the sensorium has cleared is a rare occurrence and must be considered in relation to certain constitutional factors which will be discussed later.

TABLE 1.—PRINCIPAL FEATURES OF THE NEUROLOGIC AND PSYCHOTIC SYNDROMES ENCOUNTERED IN EIGHTEEN CASES

Case No.	Neurologic Syndrome	Psychotic Syndrome
1	Oculomotor with choreiform twitches; paresthesia, left hand and involuntary movements after nine months	Insomnia, mental and motor hyperactivity of occupational type, followed by drowsiness; long period of physical and mental insufficiency, gradual improvement; after nine months full recovery (?)
2	Oculomotor; Benedikt's syndrome; residual tremors, left arm	Drowsiness; slight delirium and nocturnal restlessness, then lethargy; no residuals after six months except possibility of slight change in disposition
3	Oculomotor; Pseudoparkinsonian; no residuals	Lethargy; semistupor; disturbance in time relations; change in disposition; subjective impairment in powers of memory after seven months
4	Oculomotor; partial left hemiplegia; pseudoparkinsonian; residual slight rigidity of upper extremities	Vague fears; drowsiness; lethargy; alteration in character apparent after one year
5	Oculomotor; severe pseudoparkinsonian; thalamic facial weakness (left); after one year residual parkinsonian features	Stupor, mutism and tendency to maintain given positions; then euphoria and uncontrollable laughter; no mental residuals after one year except possibly abnormal stubbornness
6	Pseudoparkinsonian; general rigidity, mask-face, unilateral pains in limbs; no residuals	Lethargy; confusion, prolonged immobile state and gazing; tearful on stimulation; anxious ideas and a few hallucinations; recovery in ten months, but possibly more emotional than formerly
7	Myelo-encephalitis (cervical); oculomotor; pseudoparkinsonian; residual weakness and atrophy of muscles of shoulder girdle	Lethargy; delirious episode; anxious depressive mood; after eight months residual nonincapacitating depression
8	Oculomotor; occasional blurring of vision after eight months	Nocturnal insomnia with occupational delirious episodes; somnolent during the day; rapid improvement but persistence after eight months of somnolent tendency
9	Oculomotor with suggestion of cerebellar involvement, dizziness and disturbance in equilibrium; no residuals	Insomnia, hallucinatory excitement, then short, violent delirium; drowsy period and recovery in two weeks; no residuals after eight months
10	Oculomotor; slight thalamic facial weakness (left); tremors in hands; no residuals	Insomnia, then confusion, delirious ideas and fear followed by lethargy; change in disposition apparent after ten months
11	Oculomotor with suggestion of cerebellar involvement, later involuntary movements, facial weakness and parkinsonian gait; residual squint	Acute hallucinatory delirium interrupted by stupor; then a weak, irritable state followed by hyperkinetic reaction and elation with clear sensorium; no defects in memory or mental capacity; excitement continues after one year.
12	Oculomotor; tremors; pyramidal tract involvement (right); residual speech and facial tremor	Stupor and delirious excitement, then euphoria and uncontrollable laughing attacks; after six months shows poor emotional control; episodes of anger and crying
13	Oculomotor with muscular twitches in limbs; transitory Argyll Robertson pupil; no residuals	Delirium followed by lethargy; peculiar emotional reactions, inertia and low mental tension but no definite loss of mental capacity; unchanged after eleven months
14	Abrupt onset head pain; unilateral convulsion then coma; slight facial weakness; no residuals	Coma; afterward disorientation and confusion with dreamlike ideas and hallucinations; some restlessness and emotion; change apparent after three months
15	Oculomotor with meningeal symptoms; no residuals	Initial somnolence followed by hallucinatory delirium; recovery in three months with amnesic period; no residuals after one and one-half years
16	Oculomotor with left facial weakness, leg pains and sensation of twitching in tongue; physical symptoms persist after two years	Stupor, then depressive complaining attitude in reaction to physical condition; paranoid episode with hallucinations; trend not further elaborated after two years
17	Oculomotor with hemiplegia developing during stupor; residual hemiplegia	Stupor with confusion followed by suicidal depression, hallucinations and fears; peculiar emotional reactions; no definite mental deterioration; psychosis continues after fourteen months
18	Oculomotor; tremor of hands and tongue; no residuals	Stupor followed by occupational delirium; schizophrenia in individual with former tendencies in that direction; unchanged after ten months

On the other hand, we have found in a large majority of our cases peculiar emotional states which have persisted after all other mental symptoms have disappeared, so that a special type of residual seems to occur. Severe or prolonged psychotic manifestations occurred in four cases: one patient, after the stupor, developed a prolonged suicidal depression (Case 17); another, also after a stupor, developed a depressive paranoid state (Case 16); another one, after a severe delirium, showed a prolonged hyperkinetic condition with elation or irascibility (Case 11)); and in one patient a latent schizophrenia was brought to the surface with subsequent development of very marked symptoms (Case 18).

SLEEP DISTURBANCE

In fever, even occasionally in a mild febrile state, there is often undue drowsiness. In numerous organic brain conditions, for example, tumor, diffuse exudative disease, hemorrhage, to name only a few, as well as in states of so-called toxicosis resulting from metabolic disorders, there are disturbances of various degrees in the same field. It is apparent that pathologic sleep has a wide occurrence in neuropsychiatric conditions. In encephalitis such a disturbance is the most obvious mental alteration. At the beginning of the discussion, this disturbance is best put in terms of hyperfunction and hypofunction, for in this way it will be quickly brought to mind that not all patients with encephalitis show somnolence. Hypofunction of the sleep mechanism is frequently present instead.

On the hyperfunction side the excess of sleep in its mildest form amounts to a mere drowsiness. This stands at one end of the hypersomnic scale with coma at the opposite. It is serviceable to recognize the four gradations of drowsiness, lethargy, stupor and coma. In this way certain clinical distinctions are expressed and individual states more accurately described. The differences are those of degree.

Regarding the drowsy state the term defines itself. In it a certain more than usual desire to sleep and a tendency to doze does not noticeably upset the person's regimen.

When lethargy sets in, sleep of a special kind occurs. The patient sleeps more than is normal, possibly both day and night; his former occupations are necessarily curtailed. When asleep he can be very readily awakened completely. He may not return to sleep immediately, is not necessarily prostrate and may make voluntary efforts to keep busy at something. One of our patients, over a six weeks period when there was great lethargy, forced herself, under a physician's advice, to take long daily walks. This patient remained clear in her sensorium, except for a difficulty with the facts of time, indicating the kind of clouding apt to accompany that degree of sleep disturbance.

In stupor the patient is of necessity in bed and initiates nothing. There is a great reduction of activity. The patient is difficult to arouse, there is incomplete correlation between stimuli and reaction, and grave disturbance of the sensorium may be present. This may be sufficient to produce complete disorientation.

The patient in stupor may not always keep the eyes closed. The patients in Cases 5 and 6 went through an immobile state with mask-like facies, mutism and open eyes. Their difference in this may invalidate such cases for discussion under the general heading of sleep disturbance. One patient (Case 5) was not asleep, and in retrospect, he says that he understood everything and that he was unable to talk because of the rigidity of his tongue, lips and all of his vocal apparatus. These cases illustrate that the sleep disturbance so-called is only partly an alteration in the level of consciousness and that with it there is an activity disturbance. We do not think we have enough insight into gradations between variations in activity and somnolent states to warrant arbitrary limits in defining terms.

An all embracing inertness with the apparent suppression of reactions to virtually all external stimuli marks the fourth and most marked gradation, coma. Some of the writers who have used such descriptive terms as "twilight state" and "sleep of closed eyes" have plainly had in mind only the phase which is called lethargy in our nomenclature.

It has been suggested that the ease with which a lethargic patient can be aroused and the brightness and alertness then often shown are peculiar to encephalitis. It must be admitted that such a reaction is not usual in toxic-organic somnolent states, although something like it may be seen in brain tumor cases with facetiousness. On the other hand, in encephalitis it is often a question of degree or depth of the disturbance, as demonstrated by the stuporous or comatose patients who cannot be aroused, or if they can be, they show plainly dulness, lack of clearness or confusion.

When the sleep function is diminished or suppressed instead of augmented, the result is a hyposomnia, more commonly called insomnia. Climenko¹² has found insomnia as a symptom of onset. We have a similar history in three of our cases. Abrahamson¹³ describes hypersomnia as an onset reaction and this occurs without doubt in the great majority of cases. Of the hypersomnic states, drowsiness, lethargy and stupor are frequent. Coma, in contrast, is not usual. It occurs in two settings. In one, the coma develops out of the stupor and marks an

12. Climenko: *Encephalitis Lethargica*, New York M. J. **111**:531 (March 27) 1920.

13. Abrahamson: *Mental Disturbances in Lethargic Encephalitis*, J. Nerv. & Ment. Dis. **52**:193 (Sept.) 1920.

intensification of the latter, coincident with an alarming advancement of the disease in its general features. Coma in this instance is almost always terminal. The other setting in which we have found coma is distinctly opposite in nature; namely, the coma has come on at the very onset of the disease. In one instance, it came with lightning rapidity upon an individual who until that time had been in health (Case 14). A convulsion inaugurated the disease and established the coma. A case with similar onset is described by Guillaín.¹⁴ Both of these patients progressed to recovery—one with a certain alteration of character. Coma, therefore, occurring at the onset gives quite a different prognosis from that developing later in the course of the disease.

Though as mentioned before, the insomnic picture has been noted by some as a preliminary onset symptom, in the majority of cases it develops later, if at all. In these instances, hypersomnia by a gradual transition gives place to insomnia. Bremer¹⁵ says that the insomnia so frequent at the end of encephalitis is sometimes accompanied by an intense psychic overactivity. He mentions the case of a physician who had had the illness and had experienced the insomnia and hyperactive features. This physician reported that at first the insomnia was almost agreeable because of the lucidity and power of memory evocation which characterized it. Something much resembling it occurred in our Case 1.

The hypersomnia and the hyposomnia may alternate in the same patient through a definite day and night cycle, with a reversal, however, of the usual order. For example, such a patient, instead of sleeping at night, shows great restlessness and only with the coming of morning is able to sleep. The sleep which then ensues is the gripping lethargy which it is easy to interrupt but not possible to terminate. This rhythmicity in reverse order of the normal has been most frequently seen in children. Six such cases, all children, are described by Happ and Blackfan,¹⁶ who attach a diagnostic significance to this sequence. In one of our adult patients (Case 8), this day and night cycle is recorded.

It is doubtful if speculation is profitable regarding the causation of these alterations of sleep. The question of morbid sleep has always dealt with a multiplicity of factors. Regarding normal sleep, Dana¹⁷ writes, "Sleep is a biologic phenomenon which needs to be explained

14. Guillaín: *Encéphalite Lethargique Avec Crise Epileptique Initiale*, Bull. méd. **34**:983 (Nov. 6) 1920.

15. Bremer: *Formes Mentales de L'encéphalite Epidémique*, l'Encéphale **15**: 517 (Aug.) 1920.

16. Happ and Blackfan: *Insomnia Following Acute Epidemic Encephalitis in Children*, J. A. M. A. **75**:1337 (Nov. 13) 1920.

17. Dana: *Morbid Somnolence and Its Relation to the Endocrine Glands*, Med. Rec. **89**:1 (Jan. 1) 1916.

only as the waking state or the diastole and systole of the heart need to be explained. It is not forced upon the system by any special hypnotizing secretion. It is part of the inherent and rhythmic habit of living tissue." Dana points out that in sleep there is a blocking of the sensory inflow and of many association paths, and that this nerve block is caused ordinarily and in part by fatigue products acting on the synapses of the nerve cells. He follows the suggestion of Gemelli that pituitary secretion perhaps has the power of antagonizing the blocking effect of these products. If pituitary secretion is lessened, the blocking action of fatigue products is greater, and morbid somnolence occurs. Cushing and Goetsch¹⁸ had previously compared the physiology of the hibernating animal with the drowsiness and metabolic phenomena in hypopituitarism.

Inasmuch as the lesions of epidemic encephalitis so frequently focus about the cerebral peduncles with partial blocking of the aqueduct of Sylvius, the development in these cases of internal hydrocephalus can hardly be rare as pointed out by Buzzard.¹⁹ Dunlap²⁰ has found in localized midbrain edema pathologic evidence of this occlusion. The bearing of this apparently frequent pathologic condition upon the question of the somnic alteration of the patient may depend upon an association between hydrocephalus and hypopituitarism as suggested by Pollock.²¹ This is in line with previous analogous observations of Marinesco and Goldstein,²² of Bailey and Jelliffe,²³ of Cushing²⁴ and others. This association would appear to rest upon the interference with the exit from the ventricles of pituitary secretions which normally reach the subarachnoid spaces and there come in contact with the cerebral cortex.

If we follow this suggestion, we are led to account for somnolence as a pressure effect of hydrocephalus upon the pituitary gland. But hydrocephalus causes pressure elsewhere, for example, on both thalami and on the cortex as well. Even presuming that hydrocephalus is a necessary step in the development of somnolence in these cases, it

18. Cushing and Goetsch: Hibernation and the Pituitary Gland, *J. Exper. Med.* **22**:25, 1915.

19. Buzzard and Greenfield: Lethargic Encephalitis, *Brain* **42**:305, 1919.

20. Dunlap: To be published.

21. Pollock, L. J.: Hypopituitarism in Chronic Hydrocephalus, *J. A. M. A.* **64**:395 (Jan. 30) 1915.

22. Marinesco and Goldstein: Two Cases of Hydrocephalus with Generalized Adiposity, *Nouv. iconog. de la Salpêtrière* **22**:628, 1909.

23. Bailey and Jelliffe: Tumors of the Pineal Body, *Arch. Int. Med.* **8**:851 (Dec.) 1911.

24. Cushing: *The Pituitary Body and Its Disorders*, Philadelphia, J. B. Lippincott Company, 1912, p. 202.

is not clear in what manner it works—whether through effects on the pituitary gland or through pressure on the thalami, cortex or other areas. Kennedy²⁵ believes that pathologic changes based on localization of the virus in the thalami or anatomic interruption of afferent paths to the thalami explain the somnolence. Others explain it in terms of changes at the synapses of the cells, presumably of the cortex. To speak again of the pituitary gland, specific alterations in that gland distinct from secondary pressure effects may be the factor to cause somnolence. Of all these theories, none can be said to be conclusive. Perhaps the chief argument against a localized thalamic lesion being the cause of the somnolence is the type of case which shows a twenty-four-hour cycle, stupor during the day, and restlessness, often extreme, during the night.

The hyposomnia and restlessness are just as little understood as the somnolence. It would seem that endocrinal factors would most readily permit of alternating combinations of the hypersomnia and hyposomnia and of strange day and night cycles.

DELIRIUM

It is the generally accepted view that delirium, if the psychogenic variety is excluded, points to a brain condition, an injury in the broadest sense, of a transient or permanent nature. The result is an interference with mental function the clinical expression of which is the syndrome of delirium. We have already drawn attention to the resemblance of the delirium of trauma, alcohol, fever, etc., to the well-known organic types of mental disturbance and have shown the justification for regarding delirium as the acute form of the organic mental reaction.

The delirium of encephalitis presents the general characteristics of the acute organic disturbance. It occurred in fifteen of our eighteen cases in varying degrees of severity. We do not think that the three cases in which delirium was not recorded can be said to have been carefully enough observed or examined for one to say that mild transitory delirium did not occur. Delirium may appear at any time during the course of the disease. It is usually not prolonged and may in fact be limited to fleeting delirious episodes, especially at night. The cycle of nocturnal delirium and diurnal somnolence is often seen (Cases 8 and 12). One patient retrospectively remarked, "I was out of my head at night, but sensible during the day."

The most frequent sequence in our cases was for an initial drowsy or stuporous state to be followed or interrupted by delirium. Transient delirious features during a stupor may be easily overlooked. The

25. Kennedy: Epidemic Encephalitis with Stupor, *Med. Rec.* **95**:631, 1919.

patient may mutter only for a brief period, pick at the bed clothes, be harder to arouse, or the attention be more difficult to fix than usual.

In all of our cases beginning with insomnia, there followed a delirious phase which in turn was succeeded by drowsiness (Cases 9 and 10).

The content of the delirium of epidemic encephalitis, as in organic deliria generally, tends to center about habitual trains of thought and the usual daily activities of the patient, while combined hallucinations and illusionary falsification of things in the immediate environment are prominent. One patient, a former sailor, got on the roof at night and tried to climb the chimney imagining he was putting up a rigging. Another, a stevedore, imagined he was back at his work on the docks. A school teacher acted and talked as if she were in the class room with her pupils.

In some instances, the delirious content was apparently determined by disagreeable somatic sensations. One patient (Case 7) with a cervical involvement and root pains talked in a delirious way about having three arms, and at night said to his wife, "Wipe the blood off my arm—that other arm." Another patient (Case 13) who complained a great deal in the beginning of general soreness, pains and heat sensations, talked in his delirium of red pepper in the bed, of the devil's powder burning him all over, of feeling electric shocks, etc. Another patient who had a cystitis and required catheterization talked a great deal in her delirium of a criminal abortion.

In some cases, approaching more the amnesia type of reaction, the delirious content is less simple, there being more evidence of complex trends. One patient (Case 14) confused her husband with her brother and said, "I am married twice, I can't think of the other man but I have two children." She wanted to be allowed to have "two eggs" in bed with her. Once she threw off the covers and called out that the eggs (imaginary) were not touching her, that nobody was in bed with her.

It is usually found that amnesia covers more or less the delirious period as is the case in alcohol and other toxic-organic deliria. As in the latter so in encephalitis, the recollection of the delirious trains of thought may be quite well preserved, while there is partial or complete amnesia for external events. The completeness of the amnesia for happenings in the environment varies, of course, with the depth of the delirium and is apt to be "patchy," in harmony with the fluctuations of the level of consciousness. By way of contrast, it is of interest to note in this connection that in the typical psychogenic deliria, as seen for instance in hysteria, the content is usually completely suppressed, the patient having on recovery no recollection of the delirious trend.

EMOTIONAL REACTIONS

The mood alterations which accompany encephalitis have, because of their prominence and variety, always attracted a great deal of attention. Some writers have emphasized the lack of a normal emotional responsiveness, ranging from mild indifference and slight dulling to profound apathy and absence of all affective reactions. Kennedy and others have described as one of the most characteristic symptoms the "emotional stupor," in which condition the patients are totally unable to display any emotion. On the other hand, cases are reported in which the patient, when aroused from the stuporous state, is jocular and facetious. Euphoria is mentioned by some observers as occurring frequently, especially as an early symptom, while manic-like states with elation are also described. Various authors have mentioned as frequent and striking symptoms, absence of all concern and lack of any anxiety or worry about the illness. In other patients, however, there may be a prominent depressive reaction, fears and even suicidal tendencies. Finally, there seems to occur fairly often spasmodic or uncontrollable emotional reactions, crying or laughing, with or without appropriate affect.

This extremely wide range of emotional reactions indicates that the differences between individual cases are very great or that the mood varies markedly through the different stages of the disease. In giving the results of our observations, the mood changes in the early or acute stages will be considered first and then reference will be made to those which we have found to occur or persist in the later stages; in many instances, the latter might better be regarded as residuals of the disease because otherwise the patient appears to have recovered.

In the early stages, before the onset of the torpor or delirium, mood changes are not often marked. Pronounced depression does not seem to occur although some patients give in retrospect an account of a worried anxious mood during the initial period of the disease. One patient said he felt "tired and sorry and worried" about his condition. Another complained of feeling nervous and vaguely afraid. Climenko has described as a common initial symptom a euphoria with feelings of optimism, well being and unusual desire for work. None of our patients showed in the early stage such a reaction, with the possible exception of the patient in Case 1, who developed with insomnia a busy overactivity and expressed some grandiose ideas about his inventions, but there was no pronounced elation. On the other hand, we have frequently seen euphoria after a lethargic or delirious phase. In Case 5, a stuporous state was succeeded by a euphoric mood, with a great deal of laughter and excessive reaction to jokes and other emotional stimuli. In Case 12, a delirious period passed over into a euphoric condition, a gay happy

mood, with smiling and much laughter which at times seemed almost uncontrollable. Appropriate affect was, however, not lacking and no crying spells occurred. In neither of these cases was there any accompanying general excitement or impairment of judgment, so frequently seen in the euphoric states of paresis and manic-depressive insanity.

Euphoria with added features of a manic reaction does, however, occur. The mood, psychomotor excitement and speech productivity furnish a picture not distinguishable from a manic-depressive excitement. A boy, Case 11, passed through a severe delirium and made a slow and tedious improvement over a period of three months. Then there developed quite a typical mild manic state which has continued for nine months. Significant, however, of the organic foundation for this excitement is the fact that the patient recently, after complaining again of diplopia, had a convulsive seizure. Bremer reported a similar manic-like state in a boy after acute lethargic symptoms of two months' duration. The final outcome in this case was not given. Manic reactions are more apt to appear late than early in the disease and in a sense may be considered as residuals. We have not seen a typical manic state in an adult.

Mood During Stupor.—Of special interest is the emotional state during lethargic and stuporous conditions. Various observers have commented on the fact that with the onset of drowsiness and lethargy, the patient apparently becomes remarkably indifferent and apathetic, and that during the stupor the emotional responsiveness is greatly diminished or even completely suppressed. The patient's immobile face, failure to show any interest and the lack of spontaneity certainly give the impression of an affectless state. Unfortunately, very few observations have been made which include the patients' own account of their feelings during this period. As far as we are aware, no reports have been made on the behavior of the pulse, respiration, pupils or vasomotor system while the patient is subjected to emotional stimuli.

Many patients when aroused will say that they "feel all right" and have nothing to complain about except perhaps that they are sleepy and tired. In many instances, this has impressed us as being a reaction along the line of least resistance, in harmony with the great disinclination to make any exertion, mental or physical. In Case 4, this reaction is typified when the patient says retrospectively, "I was so lazy that I didn't care to have anything on my mind." On the other hand, there are patients who display emotion when specially stimulated, showing that they are capable of a certain response even though they fail to speak.

Tilney and Howe report the case of a girl who in a stupor with mutism and an expressionless face accidentally overheard a remark that

she would not get well. Immediately tears rolled down her cheeks. In our Case 6, the patient for a long time was in an immobile state with masklike face and mutism. When his wife visited him, tears would stream down his face, and often when the physician took his hand, the patient would hang on to it a little, as if he wished him to stay. On recovery, a special effort was made to get the patient to describe his feelings during the stupor. He seems to have felt very dull, was without any initiative and was, so to speak, in a negative, colorless emotional state unless he was especially stimulated in some way. Then he was apt to feel anxious and worried. As he put it, "I was careless like—indifferent—until my wife came—then I would worry about my family." (He recalled that he wept during his wife's visits.) As to his condition at other times he says, "There was really nothing on my mind." If he had to get up or was taken to the toilet he would also become anxious and feel vaguely fearful in reaction to this situation. We can report another interesting observation in this connection. It occurred in the case of a man who, while in a somnolent state in bed at home, was told that his child had been run over by an automobile. He immediately jumped up and ran to the hospital six blocks away where he then had to lie down again because of weakness and somnolence.

It is our view that the most characteristic emotional state in the lethargic or stuporous phase is the apathy which may be extreme. However, just as a patient may be aroused from the sleep so may he be aroused, at least momentarily, by strong emotional stimuli to show an appropriate affective reaction.

Late or Residual Emotional States.—Under this heading we shall discuss what appears to us to be a very significant finding, namely, the persistent emotional alterations which remain after the acute stages of encephalitis have passed, particularly after the delirium and stupor have disappeared—in some instances after convalescence or recovery has apparently been reached.

In looking over our case histories, including the after-history of those discharged from the hospitals, we were surprised to find that only two patients out of eighteen could be considered to have returned to their previous normal condition, with one other case doubtful. The striking feature in all of the fifteen patients that did not recover was a change in the emotional reactions.

Of the fourteen patients who have left the hospital, we find that only two can be said to have returned to their previous normal emotional level (Cases 9 and 15). If we leave out of consideration Case 18, a schizophrenia, and Case 1, a doubtful recovery, we have ten cases

in which there are continuing signs of some definite alteration in character or mood.

In four of these ten, the emotional change is in the direction of a depressive affect, without any lethargic tendencies remaining; in four others the change is in the direction of an emotional elevation or irascibility; in one, there is abnormal stubbornness; in another, there is an apathetic reaction, with some persistence of drowsiness.

Moreover, in the four patients still in the hospital (Cases 11, 13, 16 and 17, all of them in state hospitals) we find that the emotional disturbance stands out as a striking feature in the clinical picture, and in two cases, the affective reactions are peculiar and in a sense contradictory. The latter two cases deserve special mention as they may be looked upon as severe psychotic cases and the unusual emotional reactions have been difficult to understand.

In one case, nearly a year after the onset of encephalitis, the patient (Case 13) showed a markedly lazy, sleepy, disinterested state which was interrupted by transient singing and jig-dancing with the admission of feeling happy but without any evidence of special elation in speech or face. The usual torpor and low mental tension were easily overcome by the stimulus of questions and examination and there was then no evidence of impairment of mental ability.

The other patient (Case 17) a year and a quarter after the onset of the encephalitis, was in a depression with suicidal inclinations. The mood, however, was subject to peculiar quick changes which seemed almost contradictory—in the midst of evident anxiety and talk of being killed she could be easily induced to smile, at times even to laugh a little. This peculiar mood reaction suggested at least superficially a lack of correspondence between the affect and the ideas expressed. We do not believe, however, that we have here a dissociation of affect as occurs in dementia praecox. It is rather the quick change of mood, the lability that is misleading, for when the patient talks of being killed she appears actually afraid. The smiling seemed to be, to a considerable extent, beyond her control and was easily elicited by suggestion. She herself said, "I know it is foolish to smile." An underlying genuine affect was shown by her repeated attempts at suicide. There was only slight evidences of organic impairment of the faculties although the patient had a hemiplegia; there was a mild mental tension defect and some variability in memory and thinking capacity.

All in all, our experience tends to indicate that persistent abnormal emotional reactions form one of the most striking features, or after-effects, of the disease. The question has arisen in many cases as to whether or not a permanent damage has occurred in the emotional sphere. Peculiar mood changes are seen in patients who may otherwise be regarded as recovered.

MOTOR PHENOMENA

Many of the motor phenomena of encephalitis have an obvious psychiatric aspect and for that reason need to be considered here. We do not, of course, refer to the various paralyses, tremors, involuntary motions and tic-like reactions, although in the latter an important psychic factor cannot perhaps be excluded. The marked reduction in all voluntary motion, and also to some extent in the reflex activities, gives rise to the picture of the so-called stupor of encephalitis.

Stupor is in no sense a unitary syndrome and, as is well known, a great variety of mental and physical conditions may be involved in a stupor reaction, namely, intense apathy, retardation, blocking, overpowering terror or fear, brain torpor, organic brain disease, etc. From what we have learned regarding the sleep disturbance and the affectivity during the lethargy of encephalitis, we believe that the psychic torpor, inertia and emotional apathy are the most important mental factors in producing the stupor, while the rigidity and certain other muscular symptoms, when they occur, are the expression of an organic upper motor neuron phenomenon of the sort seen in paralysis agitans. Typical "cog-wheel" resistance is often present on passive motion.

A great many writers have referred to the appearance of symptoms during the encephalitic stupor which they call "catatonia" or "cataplexy." These terms seem to have been used synonymously without regard for their usual psychiatric meaning. Many have applied these terms, without any further qualification, to describe a stuporous patient in whom it is possible to lift a limb which the patient maintains for a time in the given position. Few if any have mentioned the length of time they have seen positions maintained. Such a symptom must be considered in relation to its setting—the patient's clearness, mental grasp, emotional state, mental content, suggestibility, neurologic status, etc.

The tendency to hold given positions seems to be most often associated with parkinsonian symptoms, especially rigidity. One patient (Case 5), when speaking retrospectively about holding positions, said he felt rigid, that he could bring his arm down but not easily, "it was too stiff."

Tilney and Howe, who have given the most complete reports on the muscular phenomena, identify the flexible hypertonus of the muscles with the parkinsonian syndrome.

Catatonia in psychiatric terminology usually carries with it the idea of negativism, although it is apparently used by some writers on encephalitis to indicate abnormal muscle tonus with a tendency to maintain given positions. We have not observed any symptoms of negativism in encephalitis cases, although it has been shown by Bonhoeffer⁷

and by Kirby²⁶ that negativistic phenomena and catatonic reactions occur frequently in toxic-infectious disorders and in emotional settings other than dementia praecox. The stupor of encephalitis as we have seen it has, however, none of the characteristics of a typical catatonic stupor such as occurs in dementia praecox. Springlike resistance, release of opposite impulses, fantastic postures, clenched fists, "schnauzkrampf" and cyanosis of extremities have not been encountered in encephalitic stupor. The rigidity of encephalitis does not suddenly disappear and no states of excitement or stereotypies, such as occur in the catatonic forms of dementia praecox, have been reported. Mutism, drooling of saliva, holding of urine, wetting and soiling the bed occur in the stupor of encephalitis and may be mistaken for negativistic reactions. To us, it has appeared more plausible to relate them to the great psychomotor inertia and emotional apathy, a view which is supported by the patients' explanations obtained after emergence from the period of inactivity or stupor.

In retrospect, patients have told us that they did not care to speak because of the effort required and the "lack of voice," and often, in addition, because of a stiffness of the tongue, throat and facial muscles. In retention of urine, it is, of course, necessary to exclude an organic alteration based on a myelitic complication.

The use of the term "catatonia" in describing the motor or muscular phenomena of encephalitis seems to us misleading.

PSYCHOTIC TRENDS AND GENERAL MENTAL CONTENT

Ideas of a specific type are, of course, not to be expected in encephalitis any more than in other organic-toxic-infectious mental disorders. One has only to recall the multiplicity of delusional ideas encountered in syphilitic encephalitis (paresis) and various infectious and toxic psychoses. One pays most attention to the setting in which the ideas occur in these psychoses. We have already discussed the general characteristics of the trends and hallucinatory experiences of the delirium of encephalitis. Transient delusions or fixed ideas that appear independently of the delirium require some mention. Cases in which these occur are the ones in which the possibility of a relation to some other type of psychosis most frequently comes into consideration. In general we may say that definitely formulated and persistent trends are infrequent in the course of an encephalitis.

Vague fears, anxious forebodings and worry about the outcome of the illness are fairly common in the early stage and often appear to express the reactive tendencies of the personality to sickness in general

26. Kirby: The Catatonic Syndrome and Its Relation to Manic-Depressive Insanity, *J. Nerv. & Ment. Dis.* **40**:694, 1913.

(Cases 4, 5 and 7). On the other hand, the striking indifference or apathy about the situation sometimes encountered seems to be a part of the general lethargic tendency in its mildest or initial manifestation (see under emotional reaction).

Excitements with expression of various trends of ideas may precede the delirium or lethargy, and these may sometimes have many of the characteristics of a manic state. Such reactions are well known in other toxic-infectious psychoses and especially in paresis. In Case 1, there was an acute onset with insomnia, busy activity, productivity of speech and expression of what seemed to be a grandiose trend, talk of inventions of perpetual motion, of a torpedo which could cross the ocean, etc. Later it was learned that the patient had long been interested in inventions and perpetual motion. An interesting feature of this case was the fact that the excitement, which only lasted a few days, occurred mainly at night, and the patient later described a state of mental hyperactivity with his mind running continually on some detail of his work, as he expressed it, "What I was thinking about would stay in my brain all night long." Although there was said to be no clouding of the sensorium at this time, one might think the whole reaction somewhat suggestive of a not fully developed nocturnal delirium with occupational activity.

In Case 15, a circumscribed paranoid trend developed after the stupor, while the patient was in a state of dissatisfaction and worry over her physical condition and the treatment she was receiving. This reaction suggests a schizophrenic episode in a person whose make-up was distinctly of a dementia praecox type.

In Case 18, a very peculiar fantastic trend of religious sexual content followed a typical encephalitic attack (lethargy and delirium). In this case, which we regard as one of dementia praecox, we find distinct evidence of a change in character and a shifting of religious point of view prior to the encephalitis. It is our view that in this case an incipient schizophrenic reaction has been intensified coincidently with the brain disease to a point at which a fully developed psychosis may now be recognized.

In Case 17, a severe and prolonged suicidal depression with paranoid ideas and hallucinations followed the acute phase of the disease. With clear orientation, she accused the physician of wishing to kill her, voices say she was a bad woman, that she "broke the Jewish temple." A very peculiar emotional reaction accompanied the depression. She smiled in the midst of talking about being killed; the patient seemed unable to control this smiling, which could also be brought out on suggestion. She herself characterized it as "foolish." An underlying

strong effect is seen in her repeated suicidal attempts. This peculiar emotional reaction seemed to be quite different in nature from the discrepancy between mood and ideational content so often seen in dementia praecox.

MENTAL GRASP, ORIENTATION AND MEMORY

We have spoken of the mental clouding during the delirium and have also touched upon questions of orientation and mental grasp during somnolent and stuporous states. Observers usually say that when patients can be aroused from sleep or even stupor, they are often remarkably clear as to their environment, etc. The degree of clearness can, however, vary a great deal even within a short space of time. We know that often a patient who appears clear during the day may be delirious at night, in fact, a patient in a lethargic or stuporous state may sink at any time to a lower level of consciousness, mutter and show delirious "dips," for a short or longer period.

Even in patients who are apparently clear when awakened, able to appreciate where they are and to recognize those about them we have found on closer examination there were evidences of interference with the mental processes and difficulty in grasping more complicated things, especially when sustained effort and thinking were required. Facts learned by rote and habitual memories may be at command, while mental operations of a more complex character are quite beyond the patient. This we interpret as a mental tension defect, such as occurs in acute organic reactions generally.

We have given examples of the ease with which a stuporous patient may be stimulated and raised to a higher level of consciousness and emotional activity. In a typical toxic-organic delirium, as for example, delirium tremens, it is, of course, well known that the patient may usually be readily influenced by questions and appropriate stimuli and be lifted to a level where he is able to give quite accurate personal data and other information acquired prior to the onset of the delirium. One of our patients, after the stupor was over and general clearness as to environment existed, took hours to compose a note of a few lines to his wife and made many mistakes in spelling. (Later after full recovery he had no trouble in performing such a task.)

We have found in our cases very little evidence that any severe or lasting impairment of orientation or memory follows the acute stages of the disease. If the brain should be permanently damaged by diffuse or coarse lesions, as is quite conceivable in encephalitis, one would expect some intellectual impairment, or dementia. We have, however, seen very few indications of a deterioration of this nature in any of our patients. We have examined carefully three patients, with severe

symptoms, who remained in state hospitals, for the purpose of determining whether or not a definite intellectual impairment could be demonstrated (Cases 13, 16 and 17).

In one of these (Case 16), there was evidence that a slight mental tension defect remained, but with attention and effort which was easily obtained, the patient showed no impairment in the fields of memory or retention.

In Case 13, the mental tension defect was more marked, with some torpor and disinclination to make mental exertion. He sometimes made mistakes in time relations and in recalling incidental occurrences. However, under the stimulus of questions, he was able to register and retain impressions very satisfactorily and to give accurate information about the remote past—showing that his memory was good.

In Case 17, there was more evidence of an impairment of the intellectual faculties, but it cannot be said to have been of severe grade. There was variability in the accuracy of her memory, and capacity for mental operations. On specific tests, with good attention, the patient could, however, do very well on the memory and retention tests although she complained of a certain feeling of difficulty in mental application.

So far as our observations go, we have not found any marked defects in the intellectual faculties as a result of encephalitis. In only one patient (Case 17) did there seem to be some impairment, this being a patient who suffered a hemiplegic attack during the stupor and subsequently developed a prolonged depression. Whether or not these severe psychotic cases will show later on, more evidences of a permanent organic type of mental impairment is a question which we leave open.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis, one must take into consideration principally: (1) toxic states with mental symptoms, (2) infections with cerebral complications, and (3) various kinds of organic brain disease with mental disturbance, including disorders of the pituitary gland.

A large array of toxic states and poisonings, lead, carbon monoxid, alcohol and some food poisonings, chiefly botulism, diabetic and uremic states of coma, may require consideration. Various febrile deliria and infectious psychoses may have to be excluded, especially acute cerebral syphilis and meningitis. Numerous organic brain diseases, trauma, tumor, etc., may figure in the differential diagnosis.

The criteria by which these conditions may be separated from encephalitis are the history of the case, the physical signs and the laboratory findings. The psychotic symptoms of encephalitis show nothing in themselves sufficiently characteristic to distinguish them from the mental reactions in other toxic-infectious-organic conditions. In them

all, the acute organic syndromes as outlined earlier in this paper may be present, in each, in various degrees of severity. A single exception to this statement is the reversal of the usual day and night sleep sequence. As far as we are aware this has not been noticed, or at least reported, in conditions other than epidemic encephalitis.

It is, of course, well known that in infectious psychoses and in various toxic-organic mental disorders delirium and restlessness are more apt to occur at night.

SUMMARY

The psychic disturbances of epidemic encephalitis present the general characteristics of an acute organic type of mental reaction, corresponding more specifically to a toxic-infectious psychosis.

In the acute stages of the disease, psychic torpor and delirium are the most frequently observed mental disturbances although other clinical pictures may be encountered, as the Korsakoff syndrome or more complex mental disorders in which various affective and trend reactions give a special cast to the psychotic disturbance.

Two types of sleep disturbance occur, hypersomnia and hyposomnia. The four gradations of the former are drowsiness, lethargy, stupor and coma. Hyposomnia is not common, but is occasionally an onset symptom and sometimes occurs at other stages of the illness. A reversal of the usual day and night sleep cycle occurs, with the result that the patient is insomniac at night and somnolent during the day. It is impossible to draw fine distinctions between degrees of somnolence and states of lowered activity (stupor without sleep). Theories regarding the causation of the disturbances of sleep are inconclusive.

A great majority of patients with encephalitis show delirium at some stage. Transient delirious features during a stupor may be easily overlooked. In encephalitis, the content of the delirium tends to center about habitual trains of thought and occupational activities, but is sometimes determined by somatic sensations. The content of the delirium is usually remembered, while amnesia of different degrees, sometimes "patchy," remains for external events.

Before the onset of lethargy or delirium, mood changes are usually not marked, although certain patients in retrospect have described a worried, anxious mood at this time; and in contrast, one patient at the onset, while insomniac and overactive, expressed grandiose ideas but was not apparently elated. After the passing of the lethargic or delirious phase, euphoria frequently arises and with it sometimes uncontrollable laughter with appropriate mood. Features of a manic reaction are sometimes added to the euphoria and furnish a picture not distinguishable from a manic-depressive excitement.

Depressive reactions in various grades of severity not accompanied by retardation, although in one case with repeated suicidal attempts, have been seen following the stuporous or delirious stage.

In the lethargic and stuporous states, there is apathy and apparent inactivity. This is chiefly a disturbance of showing affect rather than an absence of affect. Emotional response of some kind can be obtained from almost every patient, provided the stimulus is adequate. Just as a patient may be aroused from the sleep, so may he usually be aroused by strong emotional stimuli to show an appropriate affective reaction. We have encountered peculiar, and in a sense contradictory, emotional reactions in patients who still have not recovered. Some of these mood reactions suggest, at least superficially, a lack of correspondence between the affect and ideas expressed. We have not found, however, dissociation of affect, such as occurs in schizophrenia. It is rather the quick changes of mood that are apt to mislead; but with these changes, there seems to be always a corresponding change in ideational content.

In all of the unrecovered cases, there were signs of some definite alterations in character or mood. This, in a large proportion of them, constitutes the only evidence of lack of recovery. These continuing alterations in character and mood consist of depressive affects, emotional elevations, irritability, explosive reactions, stubbornness, apathy, etc.

The extremely wide range of emotional reactions encountered in epidemic encephalitis probably indicates both that the differences between individual cases are very great and that the mood varies markedly through the different stages of the disease. Our findings are at least suggestive of a lasting damage in the emotional sphere in a considerable number of all cases.

Psychic torpor and emotional apathy appear to be the most important mental factors in producing the stupor, while rigidity and certain other muscular symptoms, when present, seem rather to be the expression of a motor phenomenon of the sort seen in paralysis agitans. The tendency to maintain given positions (catalepsy) is most often, if not always, associated with parkinsonian symptoms. We have not observed any symptoms of negativism in encephalitis cases, although it is well known that negativistic phenomena do occur in toxic-infectious and benign emotional disorders, for example, in manic-depressive psychoses. In none of our cases have we seen a typical catatonic syndrome such as occurs in dementia praecox. The term "catatonia" used by various writers to describe the motor or muscular phenomena of encephalitis appears to us to be misleading.

Ideas of a specific type are not found in encephalitis any more than in other organic-toxic-infectious mental disorders. The multiplicity of

mental symptoms is well known in organic-infectious disorders, for example, paresis, in which a great diversity of clinical pictures arise in association with brain changes of a very definite and specific nature. Definitely formulated and persistent trends are infrequent in epidemic encephalitis, although our series includes two fairly clear schizophrenic reactions, one of which had begun prior to the encephalitis and in the other a latent tendency was indicated in the personality make-up.

In regard to the outcome of mental symptoms of epidemic encephalitis, we have found much evidence of persisting emotional alteration with little evidence of organic mental defects or dementia.

CASE HISTORIES

CASE 1.—Indication of a psychopathic make-up, "a book worm" interested in perpetual motion schemes. Acute onset, insomnia, headache, mental and physical overactivity; few days of excited talk of inventions and perpetual motion ideas (no delirium). Then short period of drowsiness, followed by feelings of physical insufficiency without emotional depression, oculomotor symptom at onset and some choreiform twitchings. Later peculiar feelings in left hand with some involuntary movements in arms and shoulders. Physical residuals present after nine months, full mental recovery, somewhat questionable. Complaints of weak voice and lacks complete insight.

History.—E. H., aged 27, machinist, single, was admitted to Brooklyn State Hospital, Feb. 29, 1920. His parents were both healthy; one brother stammered. The patient was born in Brooklyn. During infancy he had "marasmus." He was bright at school, finished the grammar grades at 14, was always studious and was called by the family "a book worm." He was such interested in mechanical contrivances, electricity and inventions; he worked some on perpetual motion schemes; cared less for amusement than for his work. It is said, however, that he was not seclusive and that he had many friends. During the war, he served in the Navy and was assigned to duty on a destroyer. Later he was on a submarine. After his discharge from the Navy, he worked steadily at his trade, earning \$35 a week. In August, 1919, he had influenza, was given considerable alcohol and is said to have been delirious one night. He recovered without sequelae.

Present Illness.—The last week in February, 1920, he became restless and overactive; could not sleep, would stay up nearly all night going over his drawings and mechanical inventions. He continued to work at his trade until two days before admission to the hospital. He then talked excitedly all of one night about his inventions, torpedoes, steamships and perpetual motion. When put to bed he kicked about and could not sleep. When a physician was called, the patient said he had invented a torpedo which was to go from here to Europe, that he had a scheme by which steamships could cross the ocean by means of electricity generated by perpetual motion. During this time he was oriented and did not seem confused.

He was then taken to the state hospital where he almost immediately became quieter and for four or five days was noticeably drowsy. He could, however, be easily aroused and was able to give satisfactory information about himself, showing considerable appreciation of the fact that he had been upset—"nervous breakdown" as he called it.

Examination.—On the day of admission, he showed some twitchings of the hands and irregular movements of the limbs and head. The temperature was 99.2 F. There was some drooping of both eyelids. The pupils reacted well to light. He complained of double vision and blurring of eyesight. The knee jerks were active; the plantar reflexes were diminished. Lumbar puncture revealed no increase of cells and negative globulin. The Wassermann reaction on the spinal fluid and blood was negative.

Clinical Course.—Five days after his admission, the drowsiness had disappeared, also the diplopia. He said that he felt well again; spoke of having been dizzy, of being unable to read because the letters would get bigger and bigger and then blur. He gave a good account of the onset, with insomnia, restlessness and some headache covering the week before he left home. He described his overactivity at night thus: "After working all day I would start on my blue prints when I got home—I would go right through them until 1 or 2 o'clock in the morning—then some little detail that I would think about would stay on my brain all night long."

Apparently the sensorium was not at any time clouded, and no amnesia for any period of the attack could be established. He did not think that the ideas which he expressed about perpetual motion were unusual or peculiar.

Fifteen days after admission (March 14) he was allowed to go home with his father. The diagnosis was lethargic encephalitis. He did not feel able to resume work at once. He complained of weakness, seemed a trifle dull and lacking in initiative.

Subsequent Course.—April 10, 1920, he was examined. He had not regained his strength and energy. He appreciated this and remarked, "I feel lazy, tired and sleepy—I feel like dropping off sometimes." Also he mentioned that his voice was weak, "My speech just fades away—I just mutter my words." No emotional depression accompanied this subjective weakness.

Nov. 17, 1920, he was again examined. He appeared bright and alert. Recently he had opened a shop of his own. He felt that he had entirely recovered his general strength, but still thought that his voice was not so strong as before his illness. He also complained of a disagreeable drawing sensation localized in the left hand and fingers, especially on the ulnar side of the hand and the outer three fingers. This feeling had developed recently. It was accompanied by occasional involuntary motion of the left arm and elevation of the shoulder. Examination revealed no impairment of muscular power of the hands or fingers and no disturbance in tactile sensibility.

When his psychosis was reviewed he was able to give a good account of what he passed through. He confirmed his previous statements that the onset was with insomnia and busy overactivity and fussing with his drawings and inventions. He asserted that he had not had any hallucinations during the attack. Even at the time of this examination he did not think the ideas expressed during the disturbed period were delusional. He believes perpetual motion is possible and has an idea that it may be feasible to construct an electrically driven ship with small turbines in each side which will transmit a current to a large turbine so that when the ship once gets to going it will be able to generate its own motive power indefinitely.

He recalled the diplopia and drowsiness which he felt for a few days after his restlessness and insomnia subsided.

CASE 2.—*Steady worker. Athletic interests. Cheerful. Oculolethargic type. Benedikt's syndrome. No psychotic symptoms besides moderate stupor*

and early slight transitory delirious features. Short course. Some physical residuals. No mental residuals except possible alteration of character.

Family History.—S. M., aged 18, an auto mechanic, was admitted to Bellevue Hospital, Feb. 7, 1920. His parents, two sisters and one brother are living. There was no history of nervous and mental diseases. He was born in Poland, but came to the United States in early childhood. Here he attended public school, and made ordinary progress. At 15, he went to work. As a worker he was known to be very steady. He had been also an obedient son, good to his parents and free from bad habits. He had been much interested in ball playing and, more than the average for boys of his opportunity, interested in track athletics. In disposition, he was cheerful and not moody.

Present Illness.—His illness began early in February, 1920, with dizziness and diplopia, the latter causing him to attend an eye clinic. He wore a patch over one eye for a few days and then because of sleepiness he had to go to bed. He slept more than was normal, feeling sleepy both night and day. At night, there were occasional brief periods when he was restless and probably somewhat delirious; his family said that he talked "foolish," but were unable to recall what his talk was about. After a week or ten days at home he went to the hospital by street car.

Clinical Course.—In the hospital he presented a typical lethargic state, a condition of semistupor from which he could be readily aroused. He was not negativistic. Neurologically, there had developed a Benedikt's syndrome, a complete ophthalmoplegia externa on the right and a partial left hemiplegia accompanied by hemiataxia on the same side. His temperature was normal and there was a negative serology. The lethargy was of short duration and disappeared after five or six days in the hospital. Though there is record of the "foolish talk" prior to admission, there were no delirious symptoms noted in the ward. His orientation was maintained. He was able to leave the hospital after six weeks.

Subsequent Course.—When seen six months later, it was found that he had no true amnesia for any portion of his illness though he recalled that for a short period things seemed rather "foggy." There was during the illness no feeling of anxiety, no sense of impending danger or fears of death. There was not apparently a real distress over his disabling symptoms.

He returned to work one month after leaving the hospital. He had been slightly inconvenienced by a variable unsteadiness of the left hand (which showed ataxia during the acute stage). He explained that the strength was there but sometimes he could not control it so well as previously. The disturbance was noticed at comparatively infrequent periods.

Mentally, at this time, there appeared to be an almost complete return to normal. He was alert and quick, showed nothing unusual in speech or mood. There was no alteration in his interests or desires for activity, which, as before noticed, were always considerably along athletic lines. If judged alone from interviewing him, there would seem to be almost an unnatural confidence in view of his continuing partial disability. In contrast to this, however, his relatives said that he was different now and much less jolly and goodspirited than prior to the illness. To them he showed a considerable concern regarding his disability.

CASE 3.—*Good make-up. Disease began with diplopia. Great drowsiness. Partial confusion—regarding time; clear regarding people. Never hallucinatory.*

Very good recovery after eight weeks. However, residual change in disposition with great social activity and less ambition. Subjective impairment in powers of memory.

Family History.—E. B., aged 23, bookkeeper, was admitted to Bellevue Hospital, March 15, 1920, with no history of nervous or mental diseases.

Previous History.—The patient was a young, industrious, American office worker with good habits. Her education consisted of the common grades in parochial schools, followed by night high school for three years, during which time she learned stenography. Finishing at 16, she went to work immediately. For the last two years she had worked for a firm of brokers, and as an office worker had risen to a place of importance, being in charge of the firm's cash-book and check-book.

Her make-up had in all respects been excellent. Though subject to certain depressed moods, these had never been severe nor followed by any swings toward elation. There had always been a strong interest in social affairs, parties, dancing, etc., also a fair amount of outdoor life. She was a good skater, fond of the surf, though she never taught herself to swim. Her health was always excellent and she had been a self-reliant, energetic type of person.

Present Illness.—Her illness began in February, 1920, with gastric symptoms, followed by persistent dimness of vision, diplopia on reading and much drowsiness. She slept twelve hours each night and a large part of the day time. She knew people quite well but had marked tendency to lose track of the days of the week. Though there was increasing weakness, progressive loss of weight and the great sleepiness, her physician urged that she be compelled to be outdoors a great deal. Walking was forced on her. More than once, she almost collapsed, but for six weeks this policy was persisted in. Finally on the advice of others, she became a patient in the hospital. She went to the hospital alone, making the trip by street car.

Examination.—For the first two days (only), there was a fever of 100 F. The leukocyte count was 7,600; polymorphonuclears, 68 per cent.

Neurologically, there was nystagmus, with slow oscillations to the left, and rapid to the right. There was some impairment in upward movement of the eyes with a tendency to vertical nystagmus. The left pupil was larger than the right; it was also more sluggish to light. There were no changes in the fundi. There was masklike facies. There was a suggestion of a thalamic facial on the left, also some drooping of the right angle of the mouth. There was a twitching of the upper lip—particularly on the left side. There was slight stiffness of the elbow joints. No pathologic alteration of the reflexes of the pyramidal tract signs was noted. There was normal sensory status.

Clinical Course.—There was nothing strange in her attitude or behavior in the ward, beyond a drowsiness which gradually went away after the first few weeks and a slight difficulty with facts of time. She was clear regarding people. During the nights she talked in her sleep, but when awake she expressed no strange or delusional ideas.

By the middle of May, 1920, she had made virtually a complete recovery and was discharged.

Subsequent Course.—In July, 1920, she returned to her usual office work and has been able to carry on duties presumably satisfactorily to her employers. But when interviewed in October, 1920, she complained that her "memory is very bad and not improving. I am more absent-minded than anything else." She explained the fact that she had done the same variety of work without

complaints from employers by saying, "Yes, but I make notes of everything and do not trust my memory."

Tests revealed no demonstrable defects. Memory for the remote past was strikingly good when questioned on family and personal dates (poor when dealing with school knowledge).

She repeated seven digits with rapidity and persistent accuracy, and showed no attention defect.

Regarding mood, one noticed a tendency toward elation. She remarked with considerable gusto, "I am more optimistic than I was before and I'm happier." That this mood was not obviously pathologic was shown by the fact that the sister had not noticed it. Yet when questioned, the sister admitted that the patient was going more to theaters and to dances than before her illness, that she needed no urging to join any social occasion and that she was keeping poor hours. The patient then explained, "But, I feel so restless. I want to be on the go all the time." This restlessness showed itself also in an increased attention to her clothes and to the dressing of her hair, etc.

The mother of the patient, who was also interviewed, believed that the patient since her illness had shown a poor memory. But such examples as she gave were better examples of a certain carelessness, hand in hand with her mood. The mother complained that the patient "hasn't the right ambition yet"—as she did have prior to her illness.

The patient recounted all features of her illness, and showed absolutely no amnesia. She was able to recall the dreams which caused her to talk in her sleep so persistently. "I imagined terrible things at night, but never expressed them—that accidents might happen to the family. I could see them happening."

CASE 4.—Meager education. Sociable disposition. Onset with nervousness and vague fears, then somnolence. Oculomotor symptoms, partial left hemiplegia and pseudoparkinsonian syndrome. No delirium or delusions. Short course. Following illness has shown a change in mood and lack of interests with seclusive tendencies. Slight physical residuals.

Family History.—R. S., aged 42, Hebrew, shoemaker, admitted to Bellevue Hospital, Sept. 29, 1919. The family history was negative for nervous and mental disease. He was born in Russia. His schooling was scanty and included barely two years of study. Prior to coming to the United States (at the age of 27) he had spent six years in South Africa. His work there was divided between harness-making and cobbling. For a time, he owned a small shoe-store of his own. His habits were quiet. He disliked drinking, took small interest "in women" and married at 28. As a boy he is said to have had good health and to have been active. Yet he avoided rough games and found his chief pleasure in rather excessive reading. He could skate and swim. Prior to his illness, he took a lively interest in social affairs, often played card games, etc., with a neighborhood circle. He was always known to be jolly and not given to gloomy periods of any character. In later years, he lost interest in religious affairs though previously he was taught and was inclined to be orthodox.

Present Illness.—His illness began one month before admission to the hospital with severe headache, "like neuralgia," and occasional vomiting. He was nervous and "felt afraid" in a vague way without clear-cut ideas regarding any cause for this. Although he was sleepy, he did not stay in bed. His vision was dimmed and off and on he saw double. He came to the hospital by automobile.

Examination.—On admission he was more or less somnolent with slow reaction time, although able to answer questions rationally. He showed a bilateral ptosis with inability to converge or depress the eyeballs to a normal extent. There was a slight deviation of the tongue to the left, weakness of the left side of the face, with a bilateral masklike expression and weakness of the left arm and leg. However, there were no pathologic reflexes. The sensory findings were normal, likewise the fundi. There was fever for only a few days, not rising above 101 F. The serologic examination showed 7 cells per cubic millimeter in the spinal fluid.

Clinical Course.—There was no disorientation; also no hallucinations or delusional ideas. There was continued insight. During his stay in the hospital, coarse tremors developed in the arms and hand, and when he was allowed to be up, his posture and gait showed the characteristics of paralysis agitans. The course of his illness was brief, and after six weeks he was discharged as cured.

Subsequent Course.—A year following his recovery, the patient was again seen. He was without physical residuals except for a certain slight rigidity of the upper extremities and a tendency for the hands to take the parkinsonian position. Mental examination revealed that he talked freely, had good retention and memory, entertained no abnormal ideas, in fact he made quite a natural impression. However, he said that he was now considerably more quiet than before his illness. His sister and all of his friends mention this to him and continually urge him to liven up and take more interest in social affairs. This he thought he was doing. He did not feel depressed nor worried. He took his usual interest in daily papers.

Although he got back to work one month after leaving the hospital, he found during the ensuing several months that he was unable to mix with people. He said, "During the winter I used to hide from people. I was always looking for a chance to be alone." Although shy regarding social affairs, he was not ambitionless nor indolent. There was no lessened ability to work parallel to the lessened ability and lessened desire to mix with people. Although he could think of nothing definite to cause him concern and even denied feeling sad, when asked why he was more quiet than before he said, "My mind brings more worries than it did before." No better explanation of his loss of liveliness could be drawn from him.

For the period of his illness there was no amnesia. The vague fears which he felt early in the attack did not relate to ideas of dying. Such a thought did not enter his mind. In fact, he was not at all distressed at being sick. Touching on his mental state during the course of his illness he said, "I was so lazy that I didn't want to have nothing on my mind."

CASE 5.—*Even-tempered. Optimistic. Regular habits. Efficient. Slow onset oculolethargic syndrome; later very severe pseudoparkinsonian syndrome. General rigidity. Kept given positions. Unresponsive. Stupor. Later euphoria with uncontrollable laughter. In retrospect, depressed mood during lethargic and stuporous stage. Though physical residuals (pseudoparkinsonian) are incapacitating for work, there are no demonstrable mental residuals except a possible abnormal stubbornness.*

History.—H. S., aged 32, violinist, admitted to Bellevue Hospital, Oct. 15, 1919, with a negative family history, was born in Russia, received only a poor schooling, in all about four classes, but learned to play the violin by which means he made his living. He came to the United States at the age of 23, and

over here made a fair living because he was leader of his own small group of musicians and had plenty of work to do, playing for dances, etc., on the lower East Side of New York where he lived. He was married and there were two healthy children.

He had always been steady and regular in habits, never drank, used cigarets in moderate excess, and had never had any venereal disease.

In make-up, he was even-tempered and good natured, good to his family, optimistic, never gloomy nor worried. He was not easily angered. He read the newspapers daily because he liked to keep informed. He inclined to attend the synagogue regularly, even sometimes twice a week. He was fond of social affairs, other than card games; always felt well and was not a complaining type. In 1918, he had influenza for ten days without any complications or residuals.

Present Illness.—The patient's illness began in September, 1919, with severe headache, weakness and drowsiness. His head felt as if there were wheels in it. There was no diplopia at that time and no ocular symptoms later. For three weeks he was in bed at home. He knew his wife and others, was not confused regarding time and did not express any peculiar ideas. He was able to walk to and from the lavatory until the last few days prior to admission to the hospital. It had also by that time become difficult for him to take food and almost impossible for him to talk owing to the rigidity of the tongue, lips and face. Similar rigidity was general throughout the body musculature on admission.

Examination.—In the hospital, the fever of 101 F. remained for only one day. The leukocyte count was 12,200, 69 per cent. polymorphonuclears. The blood pressure was 120; the Widal test was negative. Neurologic examination revealed no defects in the extrinsic ocular movements. The pupils were normal; the fundi negative; the facies masklike. There was an unwinking, reptilian stare. Marked muscular rigidity was noted in all extremities, and in the facial muscles, also in the tongue. Lateral movements of the lower jaw were impossible. There was a tendency to maintain the arms in given positions. There was increase in the ligamentous tone; there was no stiffness of the neck. The Kernig sign was absent. Deep reflexes were obtainable when muscular relaxation was secured. Plantar reflexes were normal. No demonstrable sensory changes were noted. The blood Wassermann reaction was negative. The spinal fluid showed 20 cells, a negative Wassermann reaction and the colloidal gold curve was 1122100000.

Clinical Course.—Four days later there was noted a midbrain tremor, affecting especially the left upper extremity, also a thalamic facial paralysis on the left.

After thirteen days in the hospital, the parkinsonian tremor was present in all extremities and still more marked in the left upper extremity. During approximately the first ten days there was marked mental torpor and drowsiness. This diminished. The facial expression brightened, although the thalamic weakness persisted. At this time the mood changed and the patient became euphoric. With this, there was uncontrollable laughter. Other patients noticed him laughing impulsively for, as they said, "two hours at a time." He felt happy at this time and the laughing, although he could not stop it at will, was in accord with his feelings. Joking about it among the patients and things in general in the ward are believed to account for the length of time it frequently lasted.

During the period in the hospital when the patient was stuporous he did not seem to understand questions, but in retrospect he says that he understood everything and that he was unable to talk because of the rigidity of his tongue, lips and all his vocal apparatus. The patient was not delirious, did not react to delusions or hallucinations, and showed no negativism.

When questioned during convalescence, the patient was able to give a good account of his trip to the hospital and later happenings. He said he "felt tired and sorry" and "worried about" his illness.

Regarding the maintenance of an imparted position he remembered that physicians put his arm in the air, and he kept it there because it was stiff. Although it was possible to bring it down, it was not so easy to do so. He "was glad to bring it down but couldn't easily—it was too stiff."

Subsequent Course.—When seen a year later (October, 1920) he showed a severe residual parkinsonian syndrome with the right hand and arm in the position assumed in paralysis agitans, variable fine tremors, masklike facies and an annoying tendency to fall forward in walking. These features had entirely precluded his return to violin playing and had kept him from work.

Mentally at that time, his attitude was normal, except for a possible stubbornness in refusing all work except violin playing. He had a good interest in affairs and quite excellent memory. He read newspapers each day and gave points concerning several matters quite accurately. He was able to repeat six digits rapidly. His difficulty in performing small tasks in arithmetic was considered in keeping with his training. He was not emotional when telling of his condition and was not unduly anxious.

CASE 6.—*Good make-up with slight indication of emotional instability. Lethargy, general rigidity and unilateral limb pains. Transitory slight confusion and delirium. Next followed prolonged immobile state, masklike face, mutism and gazing. No catalepsy or negativism; tearful on stimulation. Vague fears and anxious ideas, few hallucinations. Recovery with little amnesia. Possibly more indications of emotional instability than before illness.*

Family History.—T. R., aged 31, freight handler, admitted to Manhattan State Hospital, April 26, 1920, with a family history that was negative for nervous and mental diseases, was born in Ireland. He was healthy as a child, received instruction in the common school as far as the fifth reader and was an average pupil. He did laboring work, was industrious and steady. He came to the United States seven years ago and has since worked as a freight and express handler. In disposition, he was said to be even-tempered, friendly and sociable. He was regarded as rather "tender hearted" and easily moved to show affection. He drank whisky moderately. He was married three years ago and had two children, living and well.

In the spring of 1918, he had influenza, was in bed two weeks and recovered without sequelae. He never had any nervous or mental trouble prior to his present sickness.

Present Illness.—Early in February, 1920, he was taken sick with fever, pains in the head, neck, right shoulder and arm. He felt drowsy and "dopey" and was in bed off and on until his wife gave birth to a baby, February 22. He then made a great effort to keep up and continue at work. About the middle of March, however, he gave up and went to bed. He then felt very sleepy and heavy, and continued to have pains in his right shoulder, arm and leg. He was weak and shaky. For two or three weeks, he lay in bed almost immobile; he rarely spoke and had a peculiar blank facial expression. Occa-

sionally, he would express some vague fears and anxious ideas. He said someone was after him; he thought people in the hall talked about him but he could not understand what they said; he imagined he was to be taken away. A few days before he was sent to the hospital, he was more apprehensive and uneasy, talked of being killed by some one but did not specify by whom. He was taken to the psychopathic ward of Bellevue Hospital. He there appeared very weak, perspired profusely, and most of the time lay very quietly in bed in a rigid attitude gazing at the ceiling. He did not answer questions, although he appeared to make some effort at speech by mumbling, and he would understand and obey simple commands such as to show the tongue. There was some general stiffness when passive motion was attempted but no catalepsy.

Examination.—April 26, 1920, the patient was transferred to the Manhattan State Hospital. He then had a fever of 102 F. which gradually declined to normal in five days. The pupils reacted to light and the eye movements were free. The knee jerks were equally increased. Spinal puncture gave positive globulin, no cells and a negative Wassermann reaction.

Clinical Course.—For four months following his admission to the state hospital, the patient continued to exhibit a marked reduction in activity, but without muscular stiffness, resistance or catalepsy. He never drooled saliva, held his urine or showed other negativistic behavior. He rarely changed his posture in bed, requiring to be spoon-fed and for a time urinated and defecated without making any effort to go to the toilet or to let his wants be known. He did not appear to be drowsy during the day, as he kept his eyes open most of the time and usually gazed in one direction. The masklike facial expression was striking. When approached he would, however, give some attention, as shown by the eye movements, and on various occasions he displayed considerable emotion even though he would not speak. For instance, during his wife's visits, tears would roll down his cheeks and often he would hold on to the physician's hand and act as if he wanted him to remain. Sometimes he would mumble as if he were trying to say something. On one occasion during the early part of his hospital residence, he answered a few questions after much urging. He seemed then to be rather perplexed and unclear as to the situation. He was not certain whether he was in a hospital or not, said it was 1916 or 1920, could not tell how he came to the hospital or how long he had been here. He spoke of hearing Jewish and Italian people talking, also of hearing his wife's voice. Once in a confused way he spoke of his citizenship papers being on an empty chair by the bed. (He had been naturalized just before his illness. After recovery he said he imagined if he were known to be a citizen he would be better treated.)

During August, 1920, the patient began slowly and gradually to emerge from his inactive state. Nov. 20, 1920, he had apparently recovered and was ready to be discharged.

Subsequent Course.—He was bright and alert and capable of doing considerable work without fatigue. He had excellent insight and repeated tests failed to show any impairment of his general memory, retention, attention or mental capacity. His interests were keen, he was anxious to return to his family and his emotional reactions were adequate and stable with perhaps one exception: when talking of his wife and children, the separation from whom he felt very much, his eyes were apt to fill with tears. He said it was his nature to be affected easily and this was confirmed by his wife (see personal history).

On reviewing his illness with him it did not appear that there was any definite period of amnesia which could be defined. He recalled very well all of his movements and most of his symptoms.

He mentioned among the early symptoms a heavy, sleepy feeling and pains and stiffness, especially on the right side of the head and body (shoulder, arm and leg). Later he felt vaguely afraid that something would happen to him, that he would be taken away, perhaps killed. Hallucinations were infrequent, occasionally he heard remarks which he thought referred to him. Once he imagined he heard his brother speak, saying, "Oh, Terry, I dread the operation table." On another occasion he thought he saw his wife outside walking across the lawn (illusion?). He recalled that he lay still and inactive, gazing and not responding to questions. His explanation for this conduct was that he felt extremely weak and did not care to move or exert himself. He also said that he was vaguely fearful of attendants and other patients, thought he was disliked and thought it was best to keep quiet. He thought he realized where he was fairly soon after he came to the hospital, but he could not keep track of time. He could not give any reason for his failure to speak except that he was timid and weak, as he put it, "so sick and deadlike that I didn't care for speaking." As to his mental content during the stupor (aside from the occasional fears), he said, "There was really nothing on my mind at that time." He seemed to have felt very dull and without initiative, he was in a negative, colorless, emotional state unless he was stimulated in some way, then he was apt to feel anxious or worried. As he put it, "I was careless-like (indifferent) until my wife or my friend came—then I would worry about my family." (He was often tearful during visits.) In a somewhat similar way, if he had to get up or was taken to the toilet, he would become anxious and feel vaguely fearful under this situation. As he improved he worried a great deal about his family and his circumstances.

CASE 7.—Good make-up. Efficient. Sociable. Moderately alcoholic. Tendency to worry. Myelo-encephalitis, cervical localization with lower motor neuron signs. Also oculomotor symptoms and pseudoparkinsonian development. At onset, delirious episode. Lethargic course over eight weeks followed by depression, the latter persisting after five months in moderate nonincapacitating form with fluctuations in intensity.

Family History.—J. B., aged 35, a structural iron-worker, was admitted to Bellevue Hospital, Feb. 23, 1920, with very little information. There was no known mental or nervous disease. The patient was born in Denmark and had had only a few years of schooling. Having been made an orphan, he went to work at the age of 10 years variously as a farmhand and woodsman. Later, after emigrating to the United States, he learned the structural iron-worker's trade and in this made a good living.

Twenty years ago he had typhoid fever and subsequently gonorrhea. Syphilis was denied by name and symptom. He was married and there had been one healthy child. Though he drank considerably, he asserted that he could count on one hand the times he had been intoxicated. He was inclined to rather excessive venery, yet ceased all promiscuity on marriage. His physical strength was above the average and he described himself as "a hearty eater and a heavy worker."

He always had the reputation of being a jolly fellow—one inclined to banter on festive occasions, but he was a hard and steady worker. As a child, he was taught religious ideas scrupulously (Lutheran), and though he never at any time acquired the habit of church attendance, he always retained a religious

feeling about things so that he still each night said the prayers which he learned in childhood. Though he denied that he was ever subject to depressed periods, he told how he was always more inclined to worry about things than his wife. For example, when he was in good health and had no reason to fear unemployment, he would always feel uneasy after finishing one job until he secured another. As regards principles and character, he was a man of more than ordinary steadiness and reliability, largely owing to happiness of his domestic life. He led a tranquil, contented existence in spite of his hazardous occupation.

Present Illness.—Early in February, 1920, he began to complain of pains in his arms and shoulders, the pain being more severe on movement and pressure. After this had been present for about a week, he was compelled to quit work. At this time there was a beginning weakness of the arms. There was diplopia, then for a time some delusional ideas (see below).

Examination.—On Feb. 23, 1920, he entered Bellevue Hospital and showed the following neurologic status: The pupils were small and irregular, but reacted to light and were in accommodation. The fundi were normal. Diplopia had developed ten days before. Eye movement was well carried out; there were nystagmoid twitchings in the external lateral position. There was a suggestion of right facial weakness, tremor of the eyelids and tongue. The face was flushed and masklike. There was great weakness in both arms. The extensors were more affected than the flexors. There was weakness of the serratus magnus, greater on the right. The right scapula was winged. Triiceps reflex was not obtained; biceps reflex was exaggerated, the left more than the right. The supinator reflex was normal. There were fibrillary twitchings in the arms. The trunk was free from signs. The abdominal and cremasteric reflexes were sluggish. Knee reflexes were very active; ankle reflexes were present. There was plantar flexion on each side. In sensation tests he cooperated poorly. Nothing was made out except a possible hypesthesia to pain on the outer surface of arm.

The white blood cells were 10,400, 60 per cent. polymorphonuclears.

The spinal fluid showed 40 cells, lymphocytes, with a negative Wassermann reaction and a positive colloidal gold curve 0001210000. The incidence of the infection at the cervical enlargement was considered due possibly to his old infection (typhoid) in part, and in part to his occupation. Pseudoparkinsonian features were superimposed as well.

His temperature in the hospital was scarcely above normal—only 100 F., for a few days. While in the acute stage, he showed a somewhat lethargic state. There was no delirium in the hospital nor delusional ideas. His somber quietness in behavior matched his expressionless face.

Subsequent Course.—In retrospect we find that during his acute illness he was very anxious about himself and thought that he was going to die. His feeling was one of great depression and he was at no time euphoric.

In September, 1920, and later, it was found that residuals of the pseudoparkinsonian picture remained along with persistent great weakness and atrophy of the shoulder girdle. Because of the latter, chiefly, he had been unable to return to his well-paid work as a structural iron-worker. Instead, he was making a meager living, barely able to keep his wife and child in actual necessities, by working as a menial helper in a billiard room. In view of the great ill luck which this amounts to, he had made a fairly good adjustment. Though worried and gloomy he was not despondent or without hope. With

considerable good spirits he anticipated an ultimate complete recovery. When he was interviewed he said, "I haven't smiled for six months. Since I was in the hospital I can't be jolly—it is something I'd like to be but I can't." Later, "A joke don't seem funny to me. I see the idea all right but it don't seem funny." This was, as far as observed, never accompanied by retardation or feeling of hopelessness so characteristic of a manic-depressive depression. This sort of depression is actual and pathologic even though warranted by the economic facts already mentioned.

In this case at the onset, there were some peculiar ideas of a delirious nature for a few days. He imagined that he had three arms and that his arms were bleeding. One night, suddenly, he called to his wife, "Wipe the blood off my arm—that other arm" (meaning a third one). (It seems likely that the cervical lesion and probable coincident cervical root involvement determined the character of this delirious experience.) In retrospect he had full insight into it.

One could discover no impairment of retention or memory defect. He had a partial amnesia for the first three weeks of his illness.

CASE 8.—Make-up: quiet disposition and mild degree of intellectual subnormality. Onset with diplopia and vision difficulty, then nocturnal restlessness, insomnia and mild occupational delirium; in day time drowsy but mentally clear. Rapid improvement within few days, but after eight months showed persistence of tendency to somnolence.

History.—T. D., aged 16, press-boy and truck driver, recently a sailor in the Merchant Marine, was admitted to the Brooklyn State Hospital. His father was a chronic alcoholic; his mother was also intemperate and drifted away from her family. He was born in Brooklyn, attended school irregularly and was for a time in a children's home. At the age of 15 he had only reached 7B grade in the grammar school, indicating that he was retarded. Backwardness at school was confirmed by the patient who said that he was very slow at learning. Later at work, he was, however, quite capable, earning as high as \$30 or \$40 a week as press-boy.

In 1919, he made a trip to France as a sailor on a steamship, returning in November. Since then he had been working as truck driver.

He was noticeably quiet in disposition, but exhibited no other peculiarities of temperament. He was strong and robust, never had any serious physical illness, and had had no previous nervous or mental trouble.

Present Illness.—The onset was rapid, occurring the latter part of March, 1920, three weeks before he was admitted to the state hospital. He first developed vague symptoms of malaise and sore throat. He complained of his eyes (diplopia) and was taken to an oculist. He then stayed home from work and began to act in a peculiar way. He sat in the house all of one day playing an harmonica and falling asleep every few minutes; every now and then he would drop his instrument as he dozed off. He kept this up so long that he rubbed the skin from his lips. After this for a week he was restless and talkative by night, but as a rule quiet and drowsy throughout the day. He had fever which reached 101 F. During the night, he would run about the house singing, whistling and talking. He spoke a great deal of his trip on the sea, wanted a girl, frequently washed his hands and brushed his teeth. The doors had to be locked in order to keep him in. An occupation delirium was quite clearly indicated. Once he got out on the roof, thought he was on a ship, tried to climb the chimney and said he was putting up a rigging. He imagined he had very large hands. Once he lit matches under the bed

looking for money which was not there. During the day he gave little trouble, would stay in bed or doze in a chair, and although somnolent he could be aroused, and he then appeared quite clear mentally. The patient after convalescence said of this period, "I was out of my head at night but sensible in the day time."

About a week after he became disturbed, he was taken to the Kings County Hospital, Psychopathic Ward, March 31, 1920. There he was in a variable state of clearness, at times spoke of being on a ship, again realized he was in a hospital and that something was wrong with him. When asked how he felt, he said, "I feel dazed in the eyes—I went to a doctor and he put something in my eyes—next morning I saw double and it's been that way ever since."

Some of his replies indicated that he was either facetious or that he would fabricate on suggestion. When asked about going up on the roof he said, "Yes, that's all a fairy tale—I went on the roof to put the screen over the glass—the doctor tells us to carry up the screens and we do it." When asked if he was on a boat last night he said, "Yes, we were out on a submarine chaser."

Physical Examination.—This was made in the observation ward and revealed nothing of importance except slight external strabismus of the left eye.

Clinical Course.—From the observation ward he was sent to the Brooklyn State Hospital, Feb. 11, 1920. By this time he was improving. The nocturnal restlessness and excitement had disappeared. During the day he was drowsy and inactive when left to himself. When talked to he was attentive and responsive. He was clearly oriented, his memory for remote events was good, personal data were given correctly, and he was able to recall a great deal of what had happened during the time that he was mentally disturbed. In fact, no distinct amnesic period was established. He said that he had felt dazed at home, imagined that he had been on a ship, that his money was under the bed, etc. He recalled no auditory hallucinations, although some visual hallucinations and illusions had occurred during the acute psychotic period.

The physical examination was essentially negative. Strabismus and diplopia had disappeared. The deep and superficial reflexes were normal.

His improvement continued and March 7, 1920, twenty-five days after admission, he was discharged much improved, there still being apparently some slight dulness and sluggishness.

Subsequent Course.—When examined, Nov. 17, 1920, he had no physical residuals. The eye movements were normal. For a time after leaving the hospital, he worked as a grocery clerk, but he gave this up and at the time of examination he complained of a general weakness and said that he felt sleepy during the day. He was not working; showed no depression but was somewhat apathetic. He was mentally clear and able to give a good retrospective account of his illness. He recalled very well most of his behavior, even during the period when he appeared somewhat delirious. He had good insight; could not be said to have returned entirely to his previous condition on account of the persisting feeling of weakness and greater desire for sleep than formerly.

Dec. 8, 1920, he had returned to work and did it well although there was a tendency to fall asleep when he had an opportunity. He also complained of occasional blurring and dimness of vision.

CASE 9.—*Good make-up, efficient at work. Sudden onset with dizziness, difficulty in vision and equilibrium, inability to gage distances. Could not sleep*

and developed in a few days an excitement, heard God's voice and talked on religion. Then a violent delirious phase of short duration followed by a few days of drowsiness. Recovered in two weeks with amnesia for delirious phase. Continued well after eight months.

History.—J. R., aged 24, colored, gas-meter indexer, admitted to the Brooklyn State Hospital, April 16, 1920, with unknown family history, since he was left an orphan when an infant, was born in New York. Early development was uneventful. He went through the grammar grades and had two years at high school. He was considered bright and intelligent. He had a lively disposition and good habits, earned \$20 a week at his work. There had been no previous nervous or mental trouble.

Present Illness.—The onset was abrupt on April 4, 1920, when he complained of a peculiar dizzy feeling in the head and had immediately some difficulty in vision, stumbled and could not gage distances. He had much trouble in going up and down stairs. He felt as if objects about him were moving. He had to hold on to things for support and it was difficult for him to keep his balance. During the next few days, he complained of noises in his head and imagined he heard music, also he thought God spoke to him. He was restless, could not sleep, talked on religious subjects and said he was saved. He seemed to be confused and at times fearful; he thought something would fall on his head. He was not drowsy. He had some fever at this time. Finally he became so disturbed that on April 9, 1920, he was taken to the Kings County Psychopathic Ward.

He was then excited, noisy and violent, talked in a confused manner, at times laughed, seemed absorbed with hallucinations and paid little attention to questions. He would make such remarks as "These are my things here—my engines and stuff and a lot of goods." This excitement was followed in a few days by a drowsy condition, and April 16, 1920, he was transferred to the state hospital.

Examination.—He then showed a somnolent tendency but could be aroused easily to answer questions. He was approximately correct as to time and knew where he was, but could not tell how long he had been in the hospital (said three months when it was less than a day). He seemed to appreciate that he was sick and spoke of going out of his head. He said people tried to get blood out of his system and thought that upset him. He spoke of hearing God's voice.

Physical examination showed: temperature, 100 F.; active knee jerks; normal eye movements; pupils reacted promptly. Lumbar puncture revealed 7 cells per cubic millimeter and positive globulin. Wassermann reaction was negative on spinal fluid and blood.

Clinical Course.—He remained in a drowsy state for a few days then brightened up, became clearly oriented and had no further hallucinations.

On April 24, it was noted that he appeared to have almost recovered. There was a tendency to sleep during the day and a feeling of lightness in the head. He was able to give a good account of his previous history and present illness. He recalled getting sick on Easter Sunday in church, of being dizzy and hardly able to keep his balance. He was amnesic for the few days spent in the observation ward and did not recall his transfer to the state hospital. He could give little information about his delirious ideas or hallucinations, but recalled that his mind ran on religion and that he imagined God spoke to him.

May 2, 1920, he left the hospital fully recovered with no physical or mental residuals.

Subsequent Course.—Dec. 5, 1920, he was examined and found to be in good health both mentally and physically. He had resumed his work and was just as efficient as before his illness. There was no indication of any change of mood or disposition.

CASE 10.—*Eleven year old boy of previous timid, effeminate make-up. Oculolethargic syndrome. Slight thalamic facial weakness. At the onset, insomnia, and, for three days only, confusion with fears and excessive anxiety. Drowsiness. Subsequent to recovery noticeable increase in pugnacity and aggressiveness. School work less satisfactory for several months, probably due to lack of application. Return of former interest.*

History.—T. M., aged 11, schoolboy, admitted to Bellevue Hospital, Feb. 9, 1920, was one of three children. His parents were living. In the father and father's brothers there was a history of persistent excess in alcohol as long as it was available. The mother's family showed much superior stability.

Previous History.—This was negative except for poliomyelitis at 2½ years with complete recovery. He also had enuresis, which was still present, although in recent years less severe than formerly.

The mother complained that he never got along well with the boys, that he stayed to himself and never learned how to take his share in their sports. He had never learned to fight and the mother believed that she was "to blame for making a coward of him" because she overimpressed on his mind the idea that it was Christian-like to suffer in silence. His habit was to spend every odd minute of the time reading. When school was out he would go back home and read and his mother often wished he would stay outdoors in the air, and exercise. He occasionally did play ball with the boys in the park.

Present Illness.—In February, 1920, he developed diplopia and headache, became restless and had difficulty in sleeping. He began behaving strangely and became disoriented. He imagined that changes had been made in their house; he would wander about the house and appear surprised to find things as they were. His ideas soon related themselves to religious trends and he imagined that dire things were coming to the church. With his beads in his hands he would kneel before "stations," which were imaginary, and pray. To his mother he said, "They are going to chop down the church." At this stage he began having marked dimming of vision. How complete that was was not stated but it appears to have controlled him when he said, "I can hear my mamma, but I can't see her." He found a fur coat hanging in a closet and said that it was his mother, although he could not see her face. Other ideas related to his school affairs and he kept saying that the boys had stolen his books. The mother could not say how clear the patient kept regarding time, dates, etc., during that time.

The period of such symptoms was brief—only three days and nights. They were, in this instance, distinctly phenomena of onset.

On February 9, he was brought to Bellevue Hospital. He went to sleep immediately on admission and was aroused with much difficulty. He ran an uneventful course, never appearing extremely ill, although he was kept in bed for four weeks. His symptoms were the oculolethargic combination with slight left ptosis, diplopia, no (demonstrable) ocular limitations, slight left facial weakness, more apparent on motion, and moderate tremors in the hands

—left more than the right. Although on admission his temperature was 103-104 F., it became normal after two or three days. The laboratory findings included a spinal fluid cell count of 22 and a zero colloidal gold curve. For two weeks, the patient showed drowsiness in the hospital. There was difficulty in arousing him, but he always knew his mother when she visited him. He did no more talking regarding the ideas which have been mentioned and he at no time in the hospital displayed delirious symptoms. There was no so-called catatonia in the hospital. March 18, 1920, he was discharged from the hospital.

Subsequent Course.—When interviewed in September (six months after discharge from hospital), it was found that the patient was different in the following particulars since the illness: The outstanding difference was regarding his timidity. Instead of avoiding scraps, in his mother's opinion, he was seeking them and was taking pleasure in this new aggressiveness. The latter also might be the source of a new sort of impertinence on his part. He was no longer docile, was "bold and sassy" to both his parents. He had grown argumentative and with this had developed an excessive curiosity regarding the neighbors and even a tendency to believe they were encroaching on the rights of his own family. When this was talked over it appeared to amount to nothing more than an overpugnacious reaction to small real conflicts of interest. On the other hand, the patient was slower about doing things than previous to his illness, and according to the teacher did not learn in school so readily. This was thought to be due to lack of application. The patient himself said that he could do as well as before. During the summer he worked for a few weeks successfully in a grocery store.

In talking to the patient himself at this time, it was impossible to discover anything abnormal as regards mood, interests, ideas, etc.

Retrospectively, the patient could not recall the ideas which he had at the onset of his illness. He did recall the period of his stay in the hospital and his trip to the hospital in an automobile. He recalled his drowsiness.

In December, 1920, the patient was seen again. He was doing better at school and had more interest. He was still pugnacious with his companions and more cantankerous at home than before his illness. He was more self-assertive.

CASE 11.—Bright and well behaved boy of 9. Sudden onset with headache, vomiting and diplopia, followed by an hallucinatory delirium. This was interrupted by a somnolent period of a week, then he became again actively delirious for a few days. On clearing up he was weak and irritable for three months during which time it was noted that he had strabismus of the right eye and involuntary movements, and a parkinsonian gait. Then developed a marked hyperkinetic state with elation, distractibility, pugnacity and overproductivity of speech. Remarkably keen and alert. Orientation, memory and mental ability show no impairment. Excitement continues after lapse of a year.

History.—A. G., aged 9, schoolboy, admitted to the Manhattan State Hospital, Sept. 24, 1920, with no history of nervous or mental disease for two generations, was born in New York City, the second of four children. He was healthy as a baby, walked and began to talk at 11 months. He had whooping cough at 3 years. He perhaps cried more than the other children, but as he developed he was regarded as a good boy, easily managed, not irritable or cranky. He was fond of play with other children, was well liked by everybody.

When 7 years old, he entered school. He liked to go, learned quickly and never missed a day. His teacher made no complaints about him. He was promoted each time the class advanced.

He had had no previous sickness of a nervous or mental nature.

Present Illness.—The sickness developed abruptly about Christmas, 1919, and was ushered in by fever, headache, vomiting, diplopia, and on the second day delirium. There was "weakness in both legs"; when up, he staggered "like a drunken man." The right eye was turned. He became quieter in a few days and for about a week slept day and night. After this he was again restless and "talked out of his head." He did not seem to recognize his people, at times he swore, again he prayed, at night he spoke of seeing devils and angels. He soon quieted down, but was very weak and irritable. He slowly gained strength and at the end of three months was able to be out of bed.

He was then entirely different from his previous normal state. He exhibited an uncontrollable temper, was very active and quarrelsome. He was extremely restless, on the go continually during the day and slept little at night. He ran about the house and was constantly getting into mischief. If taken out for a walk he ran about the street, would jump on people (strangers) and kiss them; he also kissed horses.

May, 1920, he was taken to the Vanderbilt Clinic, where it was noted that he had involuntary movements of the head, shoulders and arms, left facial weakness, diplopia and external squint of the right eye. His gait was described as resembling that of paralysis agitans. A diagnosis of lethargic encephalitis was made.

June 11, 1920, because of the great difficulty in managing him at home, he was taken to Bellevue Hospital, psychopathic ward.

There it was noted that his general physical condition was good. The only positive neurologic sign was weakness of the external rectus of the right eye. There was no evidence of increased intracranial pressure. He was markedly overactive and talkative, distractible, unable to concentrate and fabricated freely. He aped and teased other patients and annoyed the nurses. He was very keen and observant, nothing that happened on the ward escaped him. He picked up information and knowledge quickly. He showed no lack of memory. He had no fixed ideas and gave no evidence of hallucinations. Sept. 5, 1920, his mother took him home; his mental condition was unimproved. She was, however, unable to manage him. He was restless, irritable and destructive. Kept the other children awake and could not be trusted alone. Ten days later (Sept. 15, 1920) he was returned to the psychopathic ward. He was then even more disturbed and excited than formerly. At times, he fought and screamed, spat at nurses and other patients, used obscene and profane language. He showed erotic behavior toward female nurses.

Sept. 24, 1920, he was transferred to the Manhattan State Hospital, Psychiatric Institute Service.

Physical Examination.—There were no positive neurologic findings except slight outward and upward deviation of the right eye. The gait and motor functions were normal. There were no involuntary movements. Lumbar puncture revealed 3 cells per cubic millimeter, negative globulin and Wassermann reaction negative.

Mental Status: There was marked hyperkinesis and diffusion of attention. He took an active interest in everything. He was quick in speech and motion, alert, distractible and mischievous; performed acrobatic stunts; delighted in teasing and annoying others, tried to take things from the pockets of older

patients, if prevented struck and fought or ran away and hid. He used profane and obscene language, ate and slept well, was cleanly in habits, but careless about the condition of his clothing which was always dishevelled.

His stream of mental activity showed overproductivity of speech and free elaboration, passing quickly from one topic to another. However, he answered questions quickly and to the point. When asked why he was in the hospital, he said, "I was sick and went to Bellevue—I am here because my mother couldn't take care of me." Why not? "I was raising the dickens." He then grabbed a stethoscope, put it to his ears and said "I am a doctor now." Took a cigaret from the examiner's pocket, stuck it in his mouth and said, "I am 13, I can smoke."

He said correctly that a social worker brought him to Ward's Island, that he came by trolley and boat. "Do you think I was swimming over—when I get out of here I am going to knock the sh— out of the social worker."

Emotional reaction was characterized by a feeling of well-being and elation, with quick changes to irritability and pugnacity. When asked how he felt, he replied, "Fine—I am having a good time here." "Are you afraid of anything?" "Who would I be afraid of? Do you want to fight (playfully)? You are a friend of mine, shake hands, we'll be pals."

There was no evidence of a delusional trend or hallucinations, no suspicions or fears.

Orientation was perfect for time, place and person.

Notwithstanding the overactivity and distractibility, it was possible to fix his attention with questions and secure cooperation with most of the tests. His memory was unimpaired except for the period of the acute illness (delirium) at home. His retention and immediate recall were excellent. He was able, for instance, to repeat correctly series of seven digits; he reproduced correctly nine out of ten word pair associations. Counting and calculation were in harmony with his education.

Subsequent Course.—Dec. 1, 1920, his condition was unchanged; the excitement continued.

December 15, the patient, after complaining again of seeing double, had a generalized epileptiform convulsion. Subsequently his mental state was unchanged.

CASE 12.—Limited education and interests. Sociable, even-tempered. Good habits. Onset with eye symptoms, then lethargy. "Cogwheel" rigidity in arms. Tremors. Pyramidal tract involvement (right). Acute delirious psychosis lasting about two months. Delirious stage replaced by pathologic euphoria, associated with uncontrollable laughing attacks. "Paretic" voice. Residual tremors. Since illness, normal mentally except for the fact that she is subject to sudden unwarranted angry spells accompanied by screaming and crying and intensification of tremors.

History.—E. P., aged 22, cigaret-maker, was admitted to Bellevue Hospital, March 11, 1920, with a negative family history. The parents and two siblings were living. She was a poorly educated factory girl. Even-tempered, sociable, with good habits and obedient to her parents. She had limited interests, was fond of moving pictures and of dancing. She was proud of the army experiences of her brother. She had had no previous serious illness.

Present Illness.—The onset was marked by eye symptoms, including diplopia, and severe head pains. After several days, drowsiness ensued and subsequently delirium. She was brought to Bellevue Hospital and owing to the delirious

state was admitted to the psychopathic ward. There she showed great restlessness and required restraint.

She was then transferred to the neurologic ward.

Examination.—On admission there, she was stuporous. Movements of the eyes to the right were limited. There was double partial ptosis, slight right facial weakness; "cogwheel" rigidity in the arms, perceptible on passive movement, also coarse tremors in the arms, with twitching movements of the facial muscles on the left side chiefly. There was more or less constant movement of the arms and legs. However, at times there was a tendency for the arms to maintain the position given them for a number of seconds. There was no hemiplegia but pyramidal tract involvement on the right. This consisted of clonus and plantar extension. The knee and Achilles' reflexes were approximately equal. There was a negative Hoffman sign. There was great respiratory distress, and breathing was accompanied by short audible inspiratory sighs, about twenty-eight to thirty to the minute.

The temperature in the hospital was 99-100 F., and at no time higher. The laboratory findings included a negative blood and negative spinal fluid Wassermann reaction, 30 cells in the spinal fluid, with positive colloidal gold series 0001232100. The spinal fluid sugar was increased.

Clinical Course.—The delirious stage lasted about eight weeks. She did not recognize persons and appeared confused. She could not cooperate, was restless and gave evidence of reacting to hallucinations. She endeavored to get from her bed and continually needed restraint.

This delirious stage gave place to a gradual return of orientation and understanding. She showed increasingly normal responses in all particulars. In respect to her mood, a real euphoria appeared. When spoken to on any conceivable subject her face broke into the broadest of smiles. Also when questioned, she said she felt very gay and happy and felt like smiling and laughing. On the other hand, she had frequent short fits of laughing which seemed to be allied to the uncontrollable laughing associated with lenticular disease. But also in these when questioned, she would say she felt happy. (There were no crying spells.) At this period the weakness of the right face was very distinctly greater during emotional expression. Because of its tremulousness and uncertainty her speaking suggested "the paretic voice." The actual distorting of speech seen in paresis was, of course, not present.

She then made steady improvement and after twelve weeks in the hospital was discharged. On leaving the hospital she showed no symptoms except a great deal of wide-spread facial tremor virtually constant and a considerable degree of the speech tremor noted above.

Subsequent Course.—When seen three months later, it was found she had been able to assume considerable housework and was returning to outside work similar to that done before her illness. She felt no incapacity for these undertakings. Her stream of thought showed no disturbances, her attitude was natural, and during the examination the mood was not unusual. It was found from her family, however, that there were alterations in mood. In contrast to a great evenness of temper prior to her illness she was now displaying anger on slight provocation at rather frequent intervals. About once a week she lost her temper, cried and screamed, did not lose consciousness and did not fall. At these times, there was great increase in the facial tremors and marked coarse jerking tremors of the arms and hand, which the patient said she could not control. These attacks were brief, lasting from only five to ten minutes. The patient appeared ashamed of them. Her parents emphasized

that they began following her illness. As regards her interests, these were meager but not different from those prior to the sickness. In a general way, moving pictures consumed all of her leisure time. There was good memory and retention and no foginess or difficulty in thinking.

In retrospect, there was a long period of complete amnesia. She recalled coming to the hospital and the wheel chair. She had no remembrances of the first ward she was in nor of the early weeks in the second ward. She said she never felt unhappy during any of her illness. She felt no apprehension regarding death and was not anxious. In retrospect, she could not recall having had delusional ideas or hallucinations, although, as noted, the latter appeared to be present at the time. When questioned again regarding her laughing attacks, she said she felt like laughing and felt truly happy. Regarding the "spells" of a different sort which she now experienced, she said that they came because she could not control her temper as well as before. That she could not keep from screaming and crying and could not control the tremors of the arms and face. She evidenced no pride in these episodes, but instead seemed to have genuine regret concerning them.

CASE 13.—Make-up: sociable, cheerful, optimistic, moderately alcoholic. Onset during third week of symptoms of infection (thought to be influenza with possibly pneumonia or pleurisy). Extreme weakness, diplopia and muscular twitchings in arms and legs. At first, drowsy during the day with restlessness and occupation delirium at night. Later, even during the day lacked clearness of mental grasp, was irritable and profane, complained of electricity and devil's powder burning him. After two months much clearer, then passed into a lazy, sleepy, disinterested state, with peculiar emotional reactions (drowsiness being interrupted by singing and jig dancing). Torpor and low mental tension; easily influenced when stimulated to effort by questions, then gave no evidence of impairment of mental ability. Unchanged after eleven months.

History.—P. G., aged 39, longshoreman, was admitted to the Manhattan State Hospital, April 2, 1920. One sister in Ireland had a psychosis from which she recovered. The patient was born in Ireland, had had no serious illness in childhood, was bright in school, but only went through the fifth reader. He came to the United States twenty years ago, and had since worked in lumber yards and on the docks. He was married seventeen years ago, his wife had had five children.

About eleven years ago, he injured his back by a fall, but made a good recovery. He drank moderately during the week and was usually intoxicated Saturday night. In make-up he was cheerful and sociable, liked to sing, joke and tell comical stories. He was fond of jig dancing at home (see later under psychosis). He had had no previous attacks of nervous or mental disorder.

Present Illness.—The patient was well until the first week in January, 1920, when he complained of pain in the left side and began to cough. He continued at work for a week and drank rather heavily to cure what was thought to be influenza. He then became very weak, complained of headaches and diplopia, felt sore all over, was feverish and had chilly sensations. He spent a week in a general hospital quarantined in the influenza-pneumonia ward. He then went back home where he was drowsy during the day but delirious and restless at night. He imagined he was back at his work on the docks, constantly tried to get out of bed, thought he saw his children in the room when they were not present. There were muscular twitchings in the arms and legs.

Examination.—On Feb. 13, 1920, he was admitted to Bellevue Hospital, medical ward. His temperature was 101 °F.; the pupils were unequal and reacted sluggishly to light, later they were noted as typically Argyll Robertson. Downward movements of the eyes were limited. The fundi were normal; the knee jerks, equal. Two lumbar punctures gave fluid contaminated with blood.

Clinical Course.—At first, he appeared very dull and slow of comprehension, hesitating in speech. The stuporous tendency alternated with periods of restlessness when he was more alert, showed some distractibility and flightiness in his talk. Often he was irritable and profane. He threw off the bed covers and exposed himself and urinated from the bed on the floor. He said someone was burning him up. During this period he was evidently quite confused. Finally he became so disturbed and unmanageable that transfer to the psychopathic ward was necessary on March 16, 1920. There he became quieter, but was confused and imperfectly oriented and expressed various peculiar ideas as shown when he said, "They put red pepper in my bed, it burns me all over. I sometimes feel electricity. They call it the devil's powder. It grows everywhere—blows in the window."

April 2, 1920, he was transferred to the Manhattan State Hospital with the diagnosis of lethargic encephalitis.

He was then very weak, unsteady on his feet, had coarse tremors of the tongue and hands and exaggerated knee jerks. The pupils were unequal and reacted sluggishly to light. Lumbar puncture repeated showed 4 cells per cubic millimeter and positive globulin. The Wassermann reaction was negative in both the blood and spinal fluid.

He was apathetic and inactive, had a blank facial expression, but answered questions quite readily, showing, however, considerable confusion. He thought it was April, 1921, the place a branch of Bellevue; he said he had been in the place four months (one day), that he had been in Bellevue one and one-half years altogether. Immediate retention was much impaired, he could not recall given names and numbers two minutes after hearing them. He was able, however, to give correctly considerable information about himself prior to his illness.

He had some appreciation that he had been sick; he once spoke of having been "a little touched in the head." He still asserted that he had been annoyed by devils who put fire in his bed, and he had felt electricity going through his body.

He improved steadily, and within two months his orientation and memory were clear. He realized that his ideas about red pepper, the devil and electricity were imaginations due to his illness. He continued to be rather sluggish, lacked energy, was careless about his personal appearance and generally indifferent. He remembered being taken sick at home, but recalled practically nothing about his residence in Bellevue or his transfer to Ward's Island. On June 28, 1920, he was allowed to go home with his wife.

Subsequent Course.—Aug. 12, 1920, he was returned to the hospital, his wife reporting that at home he had been very inactive and slept most of the time during the day, but at night he was restless, walking about, singing and talking. When taken out for a walk, he would playfully grab at women who passed. He would take fruit from a stand and say, "I'll pay you next time."

Nov. 16, 1920: After his return to the hospital he showed a peculiar lazy indifference and desire for sleep, interspersed with little outbursts of apparent elation when he sang and danced. He lounged about, stretched, yawned frequently and slept a great deal during the day. Very often, however, he would

rouse up without any special stimulation, seem rather cheerful and sing and dance jigs for a short time. Then he lapsed again into an inactive, drowsy state. The nurse described his behavior by saying, "He is either asleep or dancing." He was good-natured and docile, cleanly in his habits and expressed no peculiar ideas.

When questioned he was agreeable in manner, answered promptly but briefly, and had apparently no desire to go into any review of his case. The indifference in his general reaction was quite striking. He knew where he was, but was not clear as to time, thinking it was October, 1918, or 1919. He said he came here in February, 1917 (April, 1920); gave his home address correctly; seemed to answer at times in a haphazard way, for example, when asked if he had been sick, he answered in the negative. When asked why he was in a hospital, he said, "Oh, I was a little out of my head." When asked what the trouble was, he said, "I had pneumonia—it came from that." To the question as to how it affected him, he replied, "I was taken away unconscious from home—I remember nothing much about it." When asked about his present feelings and wishes, he said in a simple way, "I want to go home and get a decent bowl of tea." His wife stated that he was entirely indifferent about her circumstances. (She had to work and support the family.) He never asked her how she got along.

Questions elicited no trend of delusions, and he denied hallucinations. He had no complaints to make. As to the annoyances of which he formerly complained (pepper and electricity), he said those ideas were all imagination, due, he thinks, to the pains and soreness which he felt in his body.

Twice during the interview he got up and began to dance jigs, remarking, "I'll do a few steps of a hornpipe for you." When asked if he felt happy, he answered, "Sure." On another occasion when asked why he danced, he said it was to take the stiffness out of his limbs, and denied that he felt happy.

A special examination was made of his memory, retention, capacity to think and perform mental operations. The following is a summary of the results: There is a mild mental tension defect as shown by the errors he makes, e. g., as to time and place of an interview, his failure to register ordinary happenings about him, etc. When stimulated, however, by questions, he cooperates and is able to give, for the time being, good attention. His memory except for the period of the delirium is unimpaired. His immediate retention is good; he is able to repeat without error a series of nine digits, reproducing nine out of ten word-pair associations correctly, and in general retains what he makes an effort to. He does simple calculations promptly, more difficult ones he does slowly with ability to correct mistakes made. Orientation shows difficulty in keeping track of time, due probably to indifference in keeping informed and to lack of effort. He acknowledges no feeling of mental insufficiency.

As to his emotional state, he admitted a general lack of interest and loss of ambition. There was no essential depression. He claimed, when questioned about his feelings, that his only worry was about getting home. He did not insist on his discharge and was easily put off.

It was concluded that the patient showed essentially an indifference and mental torpor, a disinclination for mental effort, but no actual loss of capacity. The usual low mental tension and inertia could be fairly readily overcome when he was stimulated and brought to a higher level by questions.

CASE 14.— Alert, intelligent type. Even disposition. Sociable. Extremely acute onset with head pain, then immediately a right-sided convulsion followed

by coma lasting several days. Organic signs, facial weakness and changes in spinal fluid. No meningeal signs. Normal blood chemistry. Negative syphilitic tests. Coma succeeded by period of disorientation and confusion with dream-like ideas and hallucinations. Recovery in two weeks. Subsequent alteration in disposition. More restless. Requires more excitement.

History.—H. F., aged 21, Jewess, American born, married, housewife, admitted to New York Hospital, July 27, 1920, was one of four children. Her parents were living. The paternal grandmother was insane for a time before her death at 63, otherwise there was no history of nervous or mental disease in the family. She made a good record in common school and later in evening high school. Beginning at the age of 15 and continuing until marriage at 19, she worked as a bookkeeper in one place—a large, well-known department store. She worked with normal industry and is said to have shown more than ordinary quickness in some of the work (using an adding machine). There was a history of scarlet fever and measles in childhood. Otherwise she was never sick. Her menses had always been normal. There was no history of chorea. She was not subject to crying spells in childhood, or later, or to tantrums. She was cheerful in disposition and was always the same; she was very fond of people and of social affairs. She was not religious but attended church on holidays. Following the birth of a child in March, 1920, she developed an abscess of the breast. This caused her to become weak and nervous, and the latter showed itself chiefly when the physician, following the incision of the abscess in June, had to dress the wound daily. She would stand these ordeals very badly and would become highly upset. However, she convalesced after the abscess and on July 5 went to the country. Although she continued to be "nervous" there, in two weeks' time she gained 10 pounds in weight.

Present Illness.—We know that on July 26 she suddenly turned to her mother and said, "I've got such an awful pain in my head." Then she began crying hysterically, looked very peculiar, became unconscious and showed right-sided convulsive phenomena. That episode appears to mark the onset of the encephalitis. Five days previously, while walking with her husband, "her knees gave out" and she sank to the ground. This was transitory, and she immediately got to her feet and walked to an automobile. It is problematic whether that earlier incident should be counted as a part of the encephalitis. Following it during the five days' interval, she had absolutely no symptoms which would definitely link it with the later obvious symptoms.

At the time that she suddenly became unconscious, her right arm was drawn up and the hand strongly flexed, and her head was turned strongly to the right. These movements (apparently tonic from the account) only lasted a minute or so. The unconsciousness continued, however, and it was several days before the patient could be aroused.

In view of the later course (delirium), it is to be pointed out that the patient had not been taking any drug for sleep, that after the acute onset she was given a single hypodermic injection of morphin, and that she was received as a patient in New York Hospital in less than twenty-four hours after the onset and received no drugs there.

Physical Examination.—For two days in the hospital there was a fever of 105 F., then it was lower and reached normal after twelve days. The leukocytes were 35,000, with 83 per cent. polymorphonuclears. The chest and lungs were negative; the urine was free from pus or other pathologic findings and the blood pressure was 110. The chemistry of the blood was found to be

normal. The Wassermann reaction, in final test, was considered negative, although a weakly positive result with cholesterin antigen was first obtained. The spinal fluid on first examination showed 3 cells; later there were 20 cells. In each specimen, there was increased globulin and a positive colloidal gold curve (2233111000-1122211000) and in the second an increased sugar content (0.082 per cent.).

Neurologically, only sluggish pupils and a very slight left facial weakness (noticeable chiefly on emotion) were noted. There were no meningeal phenomena, no convulsions after the first, no change in the fundi, and no hemianopsia or pyramidal tract signs.

The patient gave variable attention. She turned a wide-eyed, childlike gaze on the examiner and displayed perplexity without fear or anxiety. There was much restlessness. She needed close watching and restraint. Both restlessness and her poor cooperation appeared due to her mood, which was one of bewilderment linked with something suggesting mild euphoria. She was disoriented in all the spheres. Her speech was quite limited in amount, somewhat but not markedly irrelevant, and not incoherent. Some days after her first visit, she failed to recognize the examiner. Hallucinations, probably illusory, were present. She thought the nurses said "Ferndale" (where she had been) over the telephone. Delirious ideas were not expressed to the examiner, but they were present, as the following shows: From her husband we find that she confused him with her brother and when they both came to see her, said she was married to the brother. Another time she said, "I am married twice, I can't think of the other man but I have two children—ain't I crazy?" On one occasion she wanted to be allowed to have two eggs in the bed with her. Then (although this request had not been granted), she threw all the bed-clothes off and called to a nearby patient to notice that the eggs were not touching her—she also said that there was nobody in bed with her.

From talks with her, following recovery, one finds that she imagined there was a small fat man under her pillow. (No idea regarding his being Lilliputian. Although he kept under her pillow he was a real-sized man.) Also she mistook the electric light bulbs for men. She would watch them "take off their heads, then their arms, and then they would disappear down the pipes" (chandelier).

A neurologic symptom, not conspicuous at the time because of her mental state, but which she described afterward, was a sight disturbance, a certain blurring of vision. Her eyesight seemed unnatural for a while, but there was no diplopia at any time. Her acute mental symptoms lasted about one week and she was able to leave the hospital after fourteen days.

Subsequent Course.—When interviewed twelve weeks after her hospital stay, it was found that her family considered her more restless than before her illness and less settled. She herself said she could not stand staying home and that she wanted excitement. She went to some theater almost every day. Otherwise, there seemed to be no change in her. She was not neglectful of her child in spite of her desire to be out. She had her usual grasp on affairs and was, for her training, properly interested in events and in her friends. The memory was good and retention was excellent. She attributed a difficulty in sleeping to the fact that at night she frequently needed to look after her young child.

CASE 15.—*Emotional instability indicated in make-up. Onset five weeks after influenza with meningeal symptoms. Pain in head and neck, vomiting and diplopia. High fever and somnolent state for a few days, followed by*

hallucinatory delirium. As sensorium cleared, she was for a short time in a depressive apprehensive state, feared she was to be tortured and killed, that an abortion was to be performed, etc. Rapid recovery in three months with amnesia period, but recollection of much of the mental content during the delirium. Remains well one and one-half years after discharge.

History.—E. G., aged 26, married, wife of bookkeeper, was admitted to Manhattan State Hospital, May 3, 1919, with a family history that was negative for nervous or mental diseases. The patient was born in New York City and graduated from grammar school at the age of 14. She was ambitious and intelligent; lively and happy in disposition, at times perhaps "too happy." She made good wages in clerical positions. She was married in January, 1918, and three months later her husband and also her brother joined the army. As a result of this separation, she felt quite downhearted and blue. She soon appeared cheerful and happy again, in fact, became rather elated, but told her friends that she did not know why she should seem so happy when in reality she was sad about the absence of her husband. (The statements regarding her make-up and the reaction to separation from her husband suggests quite strongly a cyclothymic constitution.)

Present Illness.—Jan. 25, 1919, she became ill with influenza and was treated for five weeks in Bellevue Hospital. She left the hospital apparently recovered except for general weakness and some trouble with her bladder, frequent and painful micturition, which had followed the influenza. Four days after returning home, she was again taken acutely sick with pain in the occiput, stiffness of the neck, shooting pains in the back and vomiting. Three days later, she was taken to the New York Hospital (March 8, 1919).

She was then dull and somnolent but could be aroused. Her temperature was 104 F. Lumbar puncture revealed clear fluid, 30 cells per cubic millimeter, negative Wassermann reaction, slight reduction of Fehling's solution. There were exaggerated deep reflexes, and a bilateral Kernig sign. She complained of pain in the head and neck and double vision. The urine was negative, but a week later pus cells were found in a catheterized specimen. The fever subsided and the temperature was normal the fifth day after admission. Coincident with this, the somnolent tendency was succeeded by a restless, noisy, talkative state, with evidences of a clouded sensorium. She misinterpreted her surroundings, talked disconnectedly, "used meaningless words," mumbled and "gave pseudo reminiscences." She became more disturbed and difficult to manage and made attempts to get out of her window. Her case was reported to the board of health as a case of lethargic encephalitis, complicated with psychosis. April 2, 1919, she was transferred to Bellevue Hospital, psychopathic ward.

The patient was in the Psychopathic Ward from April 2 to May 3, 1919. There she was described as being in a depressive, apprehensive state, hallucinatory, restless and disoriented. Profuse crying is said to have been a prominent symptom. Her utterances which show her confused state of mind and reaction to hallucinations are illustrated by this: "I see them passing by—there she is now—I see her doing something with a whip—she was twirling it at me—they want to whip me—listen to the marble falling down—I don't know why the people dislike me—Now what is that, who hit me on the side of the head—God tried to get rid of me." She expressed the idea that a criminal abortion was to be performed on her.

From Bellevue she was sent on May 3, 1919, to the Manhattan State Hospital. She was then in an excited, hallucinatory state. She asserted that she

saw axes, knives and flames and thought she was to be destroyed. She called out, "Fire—fire," and frightened other patients. She heard voices say she was to be tortured and killed. She denounced and cursed her husband when he visited her. The following day she was much quieter. She mistook the physician for a former acquaintance. She knew she was on Ward's Island, realized she had been sick and asked about going home.

Physical Examination.—At this time she had active knee jerks. Other wise the examination was negative for neurologic signs. There was some pulmonary congestion of both lower lobes. She was poorly nourished. The temperature for two weeks ranged from 99 to 100 F., twice going to 101.

Clinical Course.—From this time on, the mental condition rapidly improved. The fears and hallucinations disappeared, she became clearly oriented and generally cheerful in mood, with, however, some tendency to fret over being in a hospital for mental diseases, and also to cry if her husband missed coming to visit her.

By May 19, 1919, she was still very weak physically, but was regarded as having practically returned to a normal mental condition except for some emotional instability shown by weeping easily when talking of her illness and the mental distress which she went through. She seemed quite anxious to forget the thought that she had been mentally deranged, and hoped that she would transmit no taint to her offspring if she should have any. A review of her early life, personal data, school knowledge and general memory gave no evidence of any mental impairment. Her retention was excellent, thinking capacity good and insight satisfactory.

Subsequent Course.—The retrospective account of her illness showed that she was able to recall her symptoms very well until shortly after the lumbar puncture at the New York Hospital. About that time, she became confused and recalled little of what happened about her. She remembered, however, a good deal of the mental trend, including the delirious ideas and hallucinations present during the period of confusion. She thought many people were dying, it seemed that every patient put in a bed next to her died. She thought some of those who died were relatives. At times she imagined she was at the war front and saw soldiers going over the top. (Prior to her illness she had worried much about her brother and husband, both of whom were in the army.) She recalled the abortion fancy and connected it with the bladder trouble which followed the influenza and the suggestion that she might require a surgical operation. Her fear of fire was explained as being due to a dread of fire since childhood, when her mother was burned to death.

She remembered nothing of the transfer to Bellevue or her residence there. She recalled, however, coming to Ward's Island on a boat, but she thought she was on her way to Europe. After this she gradually became clear as to her whereabouts.

June 5, 1919, she left the hospital recovered from the psychosis. She spent some weeks in the country, where she rapidly gained in weight and physical strength. She then returned to her home. July, 1920, she gave birth to a healthy child.

Nov. 17, 1920, she was reported as well both mentally and physically.

CASE 16.—*Quiet, sensitive and rather seclusive make-up. Oculolethargic onset with two months of mild stupor and confusion. This was followed by general weakness, leg pains and disagreeable twitching of tongue. Prolonged depressive, complaining emotional reaction and dissatisfaction with hospital treatment. Then in a boarding house developed a paranoid trend with sus-*

pitions, delusion of poisoning and auditory hallucinations, called bad names, etc. On reentering hospital no further elaboration of paranoid ideas, but has gained no insight. Physical complaints and mild depression continue after lapse of two years.

History.—L. M., aged 42, single, seamstress, was admitted to the Brooklyn State Hospital, Feb. 17, 1920, with a family history that was negative for nervous or mental disease. She was a native of New York. She was delicate as a child and obtained a common school education. In disposition, she had always been quiet, sensitive and worrisome. She had lived a rather secluded life, making her home until recently with a maiden sister, both working as dressmakers. Seven years ago, she was successfully operated on for cancer of the right breast. There had been no previous attacks of nervous or mental trouble.

Present Illness.—About Jan. 1, 1919, she developed weakness and nausea, symptoms that were thought to indicate influenza. Within a few days, she became stuporous and slept most of the time for two months. She could be easily roused but would quickly fall asleep again. It is doubtful whether she showed any delirium, although she sometimes muttered in her lethargic state. She expressed no delusions. Her eyes felt as if they were stiff and not easily moved, and she had some difficulty in vision at this time.

Feb. 25, 1919, she was admitted to the Neurological Institute in a stuporous condition. She then had irregular, sluggish pupils and exaggerated knee jerks. Lumbar puncture revealed 6 cells per cubic millimeter. The Wassermann reaction was negative. She gradually recovered from the stupor and was discharged at the end of one month with the diagnosis of "encephalitis lethargica." Subsequently, she complained of general weakness, severe pains in the legs, and a peculiar disagreeable twitching of the tongue. Because of these symptoms, she was treated in several different hospitals. During this period, she seemed to have been quite despondent, cried a good deal, complained constantly of leg pains, and probably did not adapt herself well to hospital routine or cooperate in the treatment proposed.

While in St. Peter's Hospital, Brooklyn, during the fall of 1919, she became suspicious that the medicine was harmful and thought people were talking about her. She complained of pains and aches and wept almost continually. Because of her peculiar behavior, she was sent to the Kings County Psychopathic Ward, Nov. 29, 1919. She was then depressed, moaned and cried, spoke of the pains in her legs, a bitter taste in the mouth and a "quivering" of the tongue. She denied any paranoid trend. After two days her sister took her out and placed her in a boarding house.

She then developed a definite paranoid trend against the landlady who took care of her. She asserted that the medicine was poison, that "dope" was put in the food. She said the landlady was trying to prevent her from leaving to go to a hospital for treatment, that she wanted to keep her because she needed the board money. She said she overheard remarks made by the landlady and her son; they called her bad names and made slurring remarks, for example, "She is no good; she is a bum, a hypocrite. Her people do not care for her." She also heard something mentioned about the "sin of impurity."

Because of her trend against the landlady, her sister had to take her back to the Kings County Psychopathic Ward in February, 1920, and she was then committed to the Brooklyn State Hospital.

Physical Examination.—At this time she showed left external strabismus, moderate ptosis of the left lid, with a history of diplopia; weakness of the left side of face; right pupil larger than left, both limited and sluggish in reaction to light. There were coarse tremors of the tongue; the knee jerks were increased. Lumbar puncture revealed 4 cells per cubic millimeter, the Wassermann reaction was negative for both fluid and blood.

The mental status showed that the patient was composed and tractable. She was worried about her present situation, a little suspicious about the writing up of her history and ready to combat any suggestion that she was insane. She was clearly oriented and gave correctly her personal data. Her memory was unimpaired, except for the period of the stupor; retention was good. Simple calculations were performed promptly and accurately. She told of having influenza and sleeping two months, she said she remembered little of what happened during that time. She had subsequently suffered much from pain in the legs and from general weakness. At some of the hospitals, they did not seem to want her. The main trouble was with the landlady where she last boarded. She put something in her food, it made her vomit up bitter stuff. She also called her bad names,

Clinical Course.—Following her admission to the state hospital, the paranoid trend was not further elaborated. She continued to complain of her leg pains, could not be interested in any occupation, spent most of her time on the settees or the bed, and showed no somnolence.

Nov. 17, 1920, the patient was still in the hospital. She remained in bed because of her leg pains. She was accessible and able to give a good account of her illness and previous history. Orientation was correct, memory showed no impairment and retention was good. She was alert, attentive and responded promptly. Apperception and mental elaboration were not apparently interfered with. The emotional tone was one of mild depression and worry over her condition. This should perhaps not be considered pathologic, as she was bedridden and suffered much, so she claimed, with pains in the legs and a disagreeable twitching of her tongue. Several times during the interview, her eyes filled with tears when she spoke of her trouble and present situation.

She recalled well the onset of her sickness. She said that she had "influenza" followed by a stuporous period for two months, during which time she knew little of what went on about her. She was taken to the Neurological Institute where she improved, brightened up, but was troubled by pains in the legs and a peculiar twitching of the tongue. She was then sent to a convalescent home and later to several other hospitals. She denied that she had any suspicions or that she made any complaints of mistreatment in any of the hospitals, but admitted that in the boarding house she became suspicious and expressed the idea that the landlady was "doping her." She was dissatisfied with the place and thought the landlady was giving her quieting medicine so as to keep her from leaving; she thought the landlady needed the board money, this was the object in drugging her. She asserted that she had no suspicions or cause for complaint about her treatment since leaving the boarding house. She still believed, however, that the landlady did drug her and did make remarks about her.

There was outward and slightly upward deviation of the left eyeball. Knee jerks were equal and active. Plantar stimulation caused flexion of the toes. She complained of pain in the legs below the knee only. She lay with her feet in the position of hyperextension (suggesting foot drop). When tested, however, there was no weakness in the extensor muscles. The calf muscles

were tender to firm pressure. The nerve trunks were not abnormally sensitive. The gait was rather stiff. She could, however, walk and turn without support. There was no Romberg sign.

The tongue showed no atrophy, no marked tremor and no distinct deviation. There was slight weakness of the left side of face.

Dec. 20, 1920, her memory retention and thinking capacity were tested in detail. She showed slight mental tension defect but no impairment of the intellectual faculties. Of late, the patient had been again a little drowsy, with less interest in her surroundings. Emotional instability and depressive affect continued.

CASE 17.—Good make-up, friendly, sociable, efficient. Acute onset with fever and oculolethargic symptoms. Stupor for forty-five days, during which she developed weakness of the left side of body. Following stupor, depressive, complaining, discouraged attitude based on her illness (continued weakness and partial disability from paralysis). After eight months severe depressive psychosis with hallucinations; fear of being killed, attempts at suicide. Later peculiar emotional reaction, smiling and talking of being killed. After fourteen months continues depressed with suicidal inclinations and contradictory mood reactions. Little evidence of any organic impairment of mental faculties.

History.—D. M., aged 29, Russian Jewess, wife of a carpenter, was admitted to the Manhattan State Hospital, May 24, 1920. She had been strong and healthy as a child. She came to the United States at the age of 18. She had little opportunity to go to school, but was bright and intelligent. She was an embroidery worker and earned good wages. She was married at the age of 20, was happy and contented in her home life and had had three children. She was friendly and sociable in disposition, ready to laugh and enjoyed amusements. She adapted herself well to circumstances and was not easily discouraged. There had been no previous attacks of nervous or mental disease.

Present Illness.—The last week in September, 1919, the patient complained of headache, pain in the back and sore throat. She continued, however, to do her work until October 1, although she was feverish and somewhat restless. On October 2, she complained that something was wrong with her eyesight and that she saw double. She went to bed and then passed into a stuporous condition, during which she kept her eyes closed and could be aroused only with difficulty. Her temperature during the first few days of the stupor ranged from 103 to 104 F. On Oct. 16, 1919, she was admitted to the Willard Parker Hospital. The report from that hospital states that she was in a stuporous condition on admission, with slight rigidity in the limbs and a tendency to keep them in given positions (catalepsy). The pupils contracted. Knee jerks were sluggish, plantar reflexes were normal. No Kernig sign or rigidity of neck was elicited. There was no evidence of paralysis. The patient muttered a few words while being examined, but could not be aroused. She picked at the bed clothes.

Lumbar puncture gave a clear fluid with 20 cells per cubic millimeter. The culture was negative. The Wassermann reaction was negative for both blood and fluid.

The patient remained in a stupor with her eyes closed for about forty-five days. She could always be partially aroused by shaking, then she usually muttered, but rarely said anything which could be understood. Toward the last of November, it was noted that there was weakness on the left side (face and limbs). She also developed large bedsores.

She came out of the stupor about December 1, began to talk and quickly improved so that she could sit up. She had trouble in using the left arm and leg and complained a great deal of headache and pain in the legs. The bedsores did not heal, she became discouraged, dissatisfied and restless, and talked of being a permanent cripple. March 22, 1920, she was transferred to Bellevue Hospital (medical ward) with the diagnosis of lethargic encephalitis.

At Bellevue, she cried and complained of pain, was generally dissatisfied and begged to be taken home. She was taken home April 1, 1920. She then seemed more contented and in two weeks was able to be out of bed. She was weak, but not sleepy. She spoke about her "lazy" condition and wondered why her husband did not scold her because she was "good for nothing."

May 1, 1920, a sudden change occurred which marked the onset of pronounced psychotic symptoms. She began to talk of her relatives and husband wishing to kill her; she became agitated and restless with marked suicidal tendencies. She said she did not wish to live, she had too much trouble. She tried to jump from the window, poured boiling water on her head, drank a bottle of medicine in the hope that it was poison. She did not accuse herself of wrong doing, the main idea was that she should die on account of being sick and partially disabled.

She was returned to Bellevue Hospital and placed in the psychopathic ward. There she was anxious and apprehensive, would strike others and said the children were destroyed. The orientation was approximately correct; there were no signs of delirium. She began to show a peculiar emotional reaction. When visited by her husband, he noted that she laughed a good deal even while she talked of her children being dead. Although laughing, she did not seem happy, in fact, just the opposite.

May 24, 1920, the patient was transferred from Bellevue to the Manhattan State Hospital. She appeared very depressed, responded in a low voice, with little change of facial expression. She was oriented and asked to be killed, adding that she had poured scalding water on herself, and remarked, "I broke the Jewish temple."

Examination.—Physically, there were left hemiplegic residuals, with weakness of the face and some spasticity of the arm and leg. She walked on the ball of the left foot (talipes equinus). Knee jerks were both increased; no Babinski sign was elicited. Lumbar puncture revealed clear fluid and no cells. The Wassermann reaction was negative for both blood and fluid.

July 27, 1920, determination of a complete mental status was attempted, but the patient gave very poor cooperation. She replied indifferently to questions or showed annoyance and disinclination to answer, yet at the same time she smiled frequently. A few of her responses will indicate her attitude: Do you feel sad? "Don't speak to me." (Smiles.) Do you feel happy? "Don't speak to me—yes, I do." Treated unkindly? "No. Don't speak to me." (Smiles.) Hear voices? "By the mountain—I don't want you to speak to me."

It was not possible to ascertain anything as to the mental trend or to determine the presence or absence of hallucinations.

She was fairly clear as to time, but said she was in the Lebanon Hospital. She misidentified the other patients, also the physician. No estimate could be made of her general memory or of what she recollected of her sickness. Her stock answer was simply, "I don't know," without appearing to make any effort to give information.

Clinical Course.—During September, 1920, it was noted that in some respects the patient had improved. She was able to be up and was not so indifferent

and averse as formerly. She spoke of wanting to get well and to go home. She began to help a little with the ward work and showed that her memory was at least fair, as she had learned the names of all the patients on her ward. She was more friendly; in fact, she showed some inclination to playfulness and to tease the others. When asked the name of the place, she said "a church," but it is doubtful whether she was serious. To the physician she said, "I wish you no luck—you are going to kill me." It was still impossible to examine her satisfactorily about her illness.

Nov. 30, 1920: At several interviews lately it was found that the patient was more accessible than formerly and in some ways improved. She showed more interest, made more inquiries about things at home, helped the nurse about the ward and was attentive to some of the feebler patients. She had given up the idea that her children had been killed and did not mistake identity as formerly. There were no peculiarities of conduct.

Her general attitude and emotional state are difficult to analyze. She showed a peculiar mixture of suspicion and uneasiness, with harping, during the interview, on the idea that she was to be killed by the physician, yet she was compliant, would smile on suggestion, seemed momentarily friendly and even laughed a little. Then the mood changed quickly and she accused the physician of wanting to murder her, and seemed fearful and wanted to leave the room. At other times, she would declare, "You want to kill me," without any special show of affect; in fact, she could often be made to follow her declaration with a smile in response to the physician's smiling or joking with her.

There was a distinct depressive paranoid trend with hallucinations or misinterpretations of remarks of others. When asked why the physician or anyone should wish to kill her she said, "I insulted the whole crowd. My husband didn't pay money to you for the food." She heard this yelled out "by Christian men around the building." They also called her names as "Yiddisher colver—Yiddisher bad woman." She denied that she had done anything wrong, but at Bellevue they hollered out that she "broke the Jewish temple."

She admitted that at home she wished to die because she was nervous and sick. She thought that when she poured hot water on her head her children died. She acknowledged this was imagination.

When asked to explain why she smiled and talked of being killed she said, "I don't know myself—it is foolish to smile—I am unlucky."

She was now clear as to her surroundings but sometimes failed to give the date accurately. In general, her memory was good but there was some evidence of a mild mental tension defect as shown by the variability of the accuracy with which she recalled and correlated past events at different examinations. For example, on one occasion she made several errors in giving the birthdays of her children and was a little mixed up about the date of her marriage, whereas at another interview she gave these data correctly. When she could be induced to make an effort she showed good immediate retention. She made some mistakes in mental calculations but could correct these. On the whole, she did quite well. She herself, however, felt some loss of mental capacity, which was expressed by saying that she got mixed up easily and could not count as quickly as before her illness. (Formerly she had a very good head for business.)

In conclusion, it can be said that there were only slight evidences of an organic impairment of the faculties; a mild mental tension defect with some variability in capacity, but ability to correct any errors made. Retention was

good, when cooperation and interest were secured. A most striking symptom was the peculiar mood reaction which superficially at least suggested a lack of correspondence between the affect and the ideas expressed. We do not believe, however, this reaction was similar to that of dementia praecox. It was rather the quick change of mood that is misleading, and when the patient talked of being killed she seemed actually afraid. The smiling, however, seemed beyond her control and was certainly easily elicited on suggestion. She herself had characterized the reaction by saying, "I know it is foolish to smile." Also the underlying genuine affect was shown by her repeated attempts at suicide.

Dec. 7, 1920: The patient recently made another serious attempt at suicide by hanging. This was preceded by exacerbation of the depression and fear of being killed.

CASE 18.—Well educated school teacher. Social, athletic, intellectual type, probably over-religious and superficial in interests. Rejection of marriage on grounds directly dependent on this religious side of her character. Later regret and increasing bitterness. Began to turn against religion. Developed ideas concerning injustice of God. No peculiarities of conduct. Continued efficient at her work. Epidemic encephalitis for twelve weeks. Stupor and occupation delirium. Good recovery. Since, strange beliefs concerning the magical and remarkable results of her illness. Elaboration of lengthy religious ideas including the belief that the second Christ, to come, is to be a physician. Also believes law should be passed to make birth of children illegal. Has prepared manuscript setting forth views against God, whom she regards as a murderer and perpetrator of evil. Does not express these ideas openly in ordinary conversation, but is easily drawn out to discuss them. Continues efficient at teaching. Case considered schizophrenia, intensified by the recent brain disease in an individual with former tendencies in that direction.

History.—L. B., aged 31, teacher (domestic science), was admitted to St. Luke's Hospital Jan. 20, 1920. She was one of a large family; her parents were living. There was no history of nervous or mental disease in the family. She had had measles, chickenpox, scarlet fever and whooping cough in childhood, and at 23 otitis media, complicated by a mastoiditis, and soon afterward pneumonia and empyema, from all of which she is said to have made a good recovery. She was brought up in a substantial rural community, in a large family, where religious and, to some degree, cultural things were emphasized. In make-up, she was described as having always been both social and athletic. She was especially fond of, and clever at, winter sports. Although very much interested in intellectual matters, she was not bookish. Instead she was fond of many people and popular among them. She continually improved her knowledge for her teaching, usually taking a summer course each year. Her friend said she was known to be a good teacher. She was clever at making things, being "generally handy," yet she never took as great pride and interest in such accomplishments as her skill would have warranted. This appears to indicate a certain superficiality of interest.

A few years prior to her illness, she became acquainted with a young man of less education than herself, who, however, was making a good living in a business. Though "going straight" at the time, he had a record for excessive drinking and "came of a poor inheritance" (inasmuch as his parents had been unable to live together). He was not at all religious and was inclined to be a ready spender of money easily made. The patient, when it came to the point of a choice between an engagement or breaking the friendship, chose

the latter because of the man's "inheritance." This her friend said she did because of her family, and yet she did not actually counsel with them about it. And also as soon as she had made the decision, she became very regretful of her choice. Incidentally, the man very shortly was out of her reach. A considerable bitterness toward life then developed, and although the friend could think of no definite things which the patient said at that time, she said that "stray remarks," beginning then, showed that the patient at that time began to turn against religion and against the ideas which she had formerly fervently held.

Present Illness.—Jan 20, 1920, she entered St. Luke's Hospital, complaining of dizziness, nausea, vomiting and diplopia, and with the following history: Eight days before admission, she awoke with a dizzy feeling. She vomited a moderate amount, three times. The nausea was relieved, but the dizziness kept up. She taught school that day. The next day she awoke with diplopia, which caused more dizziness and nausea. She got through that day's work by keeping one eye closed all the time. Then she spent from the fifth to the eighth days of her illness at home in bed "trying to sleep the trouble away."

Examination.—In the hospital this showed slight intention tremor of the hands; tremor of the tongue on extension and no ataxia in finger to nose tests. There was no Romberg sign and no paralyses. Biceps, triceps, wrist, knee and Achilles' reflexes were present, equal and normal. No Babinski or Kernig sign was elicited. There was pseudo ankle clonus on both sides. The gait was that of weakness. On admission the fever was 103 F. After eight days it returned to normal, then rose again to 101 on the thirty-third day, and for a six or seven-day period subsequently was subnormal, even 96.6 on three occasions and once 96. The spinal fluid showed 21 cells, sterile culture, a negative Wassermann reaction and a colloidal gold curve of 122210000. The leukocytes were 15,700, 76 per cent. polymorphonuclears.

Clinical Course.—The following notes show her condition in the hospital. On the second day, "the patient rouses when spoken to, otherwise appears to sleep, mumbles and talks to herself." "There is general resistance to passive motion in arms and legs." Two days later, "still shows catatonia. Talks rationally when aroused." The day after the temperature became normal (and for six days it had been only 100) the note was made, "restless and talkative during night and today. She was delirious, did not know visitors and persons of the hospital and a great deal of the time talked to imaginary people as if she were at school and teaching." Her delirium was almost entirely occupational. There was a gradual subsidence of the symptomatology and after eleven weeks in the hospital she was discharged, having reached a safely convalescent condition. She went to her home in Pennsylvania by train (wheeled chair).

Subsequent Course.—The great interest in this case lies in the mental condition which the patient was found to show when interviewed after an interval of six months (October, 1920). The most striking features were an elevation of mood and an unusual productivity of speech, with the expression of a peculiar trend of ideas. The elation showed itself not alone in the content of her thought and by a great volubility, not accompanied by a flight of ideas, but also by an unusual and somewhat indecorous abandon of attitude.

She said, "I have no longer the power to suffer. I have no tears." Her speech was rapid, fluent and insistent. She brooked interruption but preferred

to keep talking. She evidenced considerable quickness of thought. Her speech was relevant—to a very slight extent incoherent. At least in regard to a creed which will be discussed later, it was ecstatic and hazy. Frequently she reverted to expressions telling how happy she now was. She was not distractible.

She said that the sleeping sickness had worked a marvelous change in her, that it had made her over, that before it her great sin had been worry and that the illness has taken "worry" from her. In fact, she remarked, "My worry center is dead." She believed that the lumbar punctures in the hospital wrought her magical cure. She knew that the encephalitis took sin (worry) from her and asked what rôle the physician had in it and also which physician it was. She expressed the feeling that there must be many people with worry as their sin, who, if they could only have the "sleeping sickness," would be made well. She felt it to be her mission (though she did not emphasize the crusade idea at all) to see that some physician found these people and inoculated them. Along with these ideas, she had formulated her antireligious ideas and recited them as her creed. This was a series of long, high-sounding phrases of religious coloring, of great length, pronounced with astonishing rapidity, clearness of enunciation and earnest fervor. Later, much the same, in less studied form, was written down and reads as follows: "My faith, in brief, is as follows: I don't believe in the existence of a God, hence prayer is futile. I don't believe in a life beyond the grave, death ends all. I don't believe in the Bible. It is the most inconsistent book that was ever written. I am happy in this belief, for I do believe, and firmly, that God was human, not divine. Possessing superhuman power, he made this wonderful earth and sky and sea which are the foundations of Heaven. Then he inhabited it with beings but left us, their outgrowth, living in Hell, for he died before his work was perfected. . . . Now I believe just as firmly in the coming of a second Christ who will perfect Creation. As the work that needs perfecting is of a medical nature, that second Christ will be a doctor, also human but exhibiting supernatural powers." She completely filled 109 pages of notebook stationery with a discussion of this topic. It is presumably an autobiographic account of her religious life, but both at the beginning and at the end, and repeated innumerable times in between, these ideas are put down along with unnumbered tirades against the injustice of "the God whom she was taught to worship. Not only is he a murderer of millions, but he is millions and millions times worse than a murderer when he hands down to innocent children the sins of the fathers." Also "when the kaiser claimed to be in league with God during the World War, were you surprised? Not I, for I believe the proverb, "Birds of a feather flock together.'" Later, on a different theme, "As the world is today, children are brought into it by the devil passion. I want to live to see the day when this powerful devil will be knocked out of existence, and common sense, the Christ, will prevail . . . until that time comes I would like to see a law passed forbidding marriages and births, a law requiring the segregation of the sexes if necessary . . . I suggest that man step in and bring it (this world) to an end."

The patient is said not to mention these ideas about God unless they are brought up. Her ideas regarding her magical cure from worry are more superficial. And she has made a point of quizzing physicians and others, not concealing her more than strange faith in the miracle the encephalitis worked in her.

Her friend believes that she is more composed and more contented than prior to her illness and more inclined than before to stay home quietly evenings and read.

There was no impairment of memory or retention. She was teaching successfully. She showed poor judgment regarding many of her present ideas. On the other hand, she manifested complete insight into the delirious experiences in the hospital. There was an amnesic period during the first part of her illness in the hospital. Then she told, with good insight, of such delusions as the following which she had: The occupational delirium has already been mentioned. She mistook the visiting rector for St. Peter. She knew she was no longer on earth, and knew that she was in Heaven. This gave her a feeling of great joy and satisfaction. She did not understand why they should take her to "Jersey" from the hospital. She was puzzled by the idea of there being a "Jersey" in Heaven. She said it took her two weeks at home in Pennsylvania to realize the true state of affairs. "After two weeks I suddenly realized that I was not in Heaven but back in this Hell of a world again."

In her account, she wrote, "In the hospital I thought I was being prepared for Heaven. Every body massage, every salt water bath, every alcohol rub, every visit of the doctor, were all done in preparation for my Heavenly home."

This 109 page article is entitled "Praise God and Stop Worshiping the Devil, Written by a Victim of the Devil and Dedicated to Those Who Need It Most, viz., Ministers, Priests, Rabbis, Missionaries, etc."

More recently she has complained of a catarrhal condition of her nose and throat and has seen several physicians for this, none of whom has been able to find anything wrong. At the same time, she complains of her saliva and considers that it is thickened and has turned white. She spends about thirty minutes each morning clearing out her nose and throat, and at later intervals during the day goes through similar prolonged procedures for this purpose. Her roommate complains that she is continually spitting in an annoying fashion. She has shown no tendency to violate usual proprieties regarding this. Her friend considers these recent ideas on this subject rather "foolish." A less intimate girl friend of the patient, having in mind the religious disbeliefs and ideas concerning her rejuvenation by means of her sickness, made no greater comment than, "My, she has a weird philosophy." In other words, there is casually observed very little unusual behavior. It is impossible to establish that she is abnormally secretive, suspicious, etc.

HISTOPATHOLOGIC FINDINGS IN A CASE OF SUPERIOR AND INFERIOR POLIENCEPHA- LITIS WITH REMARKS ON THE CEREBROSPINAL FLUID *

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REPORT OF CASE

The patient, a man, 21 years of age, was admitted on Aug. 20, 1920, to the neurologic service of Cook County Hospital complaining of headache and vomiting, with inability to swallow and to judge distance.

Previous History.—There was nothing abnormal noticed in the history of the patient's childhood except poor vision and "inability to judge distance." At the age of 9 he went to school, but "could not learn fast" because of defective vision. He was infantile in his desires and preferred to play with babies and young children.

About six months before admission to the hospital he became dizzy and fell downstairs. He was picked up uninjured, but remembered nothing about the accident.

He began to complain of his present symptoms in the early part of July, 1920. The most distressing symptom was inability to swallow, which had been present for about six weeks. For two days after admission the patient was able to swallow only small amounts of liquids. During the same period of time speech became quite indistinct and almost unintelligible.

Headaches had been present for the last two years, mostly frontal in distribution. They had been coming in attacks which lasted for days or weeks and then spontaneously disappeared for two or three weeks. For the past six weeks vomiting without nausea would set in after each meal. During the six weeks prior to admission there was a marked increase in the strabismus which had been present, in a mild form, since birth.

Previous Illnesses.—Measles, whooping cough, mumps; he had had no operations and had received no injuries.

Family History.—The patient had five brothers and one sister living and well. One sister was not bright mentally; otherwise the family history was negative.

Examination.—The appearance of the patient was that of a not well-nourished boy, dull expression and defective mentality. He gave inadequate answers even to the simplest questions, though he was well oriented as to time and surroundings. There were no visible paralyses of the spinal nerves, but almost all the

* From the pathology laboratories of Cook County Hospital and Illinois State Psychopathic Institute.

* Read at a joint meeting of the Chicago Neurological and Ophthalmological Societies, Chicago, Dec. 16, 1920.

cranial nerves were involved. Thus, the eyelids were drooping, especially the left; the eyeballs were almost totally immovable, only slight movements up and down being possible. The palpebral fissures were greatly narrowed, especially the left, which was represented by a narrow slit. The facial muscles were immobile, wrinkling of the forehead, widening of the mouth and puckering of the lips being almost impossible. The mastication muscles were markedly weak, the uvula and soft palate immobile, and swallowing even of small amounts of water was greatly impaired. In fact, the patient did not even attempt to swallow, as the food would "stick in the throat" and the liquids regurgitate through the nose. The tongue could not be protruded beyond the teeth; it showed twitchings and some atrophy but no deviation.

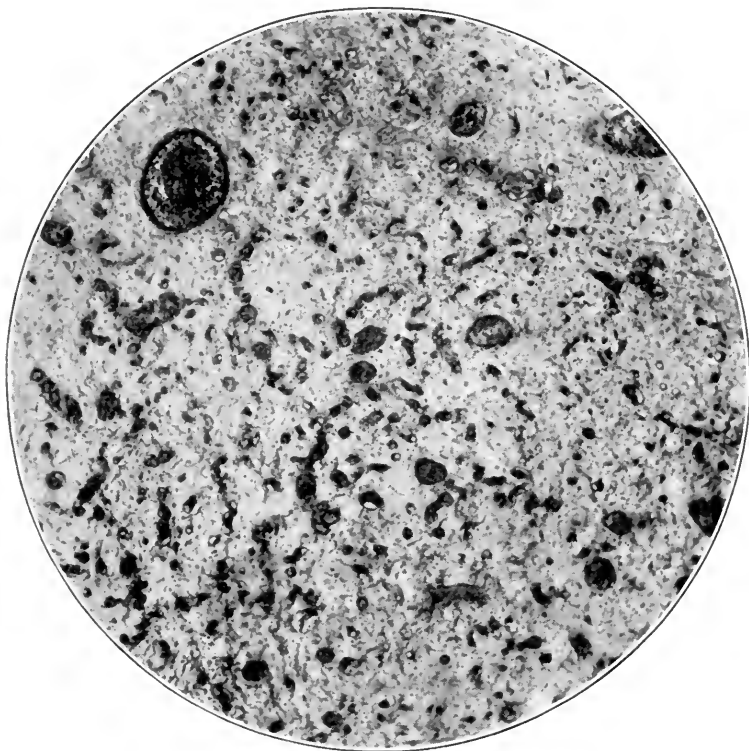


Fig. 1.—Tegmental portion of the pons. The entire area is covered by a great number of vessels and capillaries, represented in the illustration by dark, round bodies of various sizes. Some are so small that they can be seen only with the help of a hand lens as minute capillaries. The glia is vacuolated and covered by glia nuclei, ganglion cells and other structures photographed separately. Alzheimer-Mann stain; $\times 65$.

Reflexes: Both pupils reacted to light; the corneal, conjunctival and pharyngeal reflexes were greatly diminished; the triceps, knee and ankle jerks were weak; the abdominal and plantar reflexes were normal; Babinski sign and ankle clonus were absent. Sensibility was normal. The muscle power in the extremities was fair; the deltoid, supraspinati and infraspinati and pectoral

muscles were somewhat atrophied. The heart was slightly enlarged, the right limits reaching the right border of the sternum, the left limits reaching one inch beyond the left of the nipple line; the apex beat was palpable in the fifth intercostal space; there was a distinct systolic murmur. Examination of the chest revealed scattered râles on the right side, dullness over the apexes with bronchial breathing and a friction rub over the lower right lobe. Respiration was 40, the pulse rate 84 and the temperature 99 F. (by rectum).

The abdominal organs showed no abnormalities. The spinal fluid was under normal tension, colorless, with 5 lymphocytes per cubic millimeter and a negative Nonne test. More detailed examination of the patient's mental and physical condition was impossible as he died suddenly two days after admission.

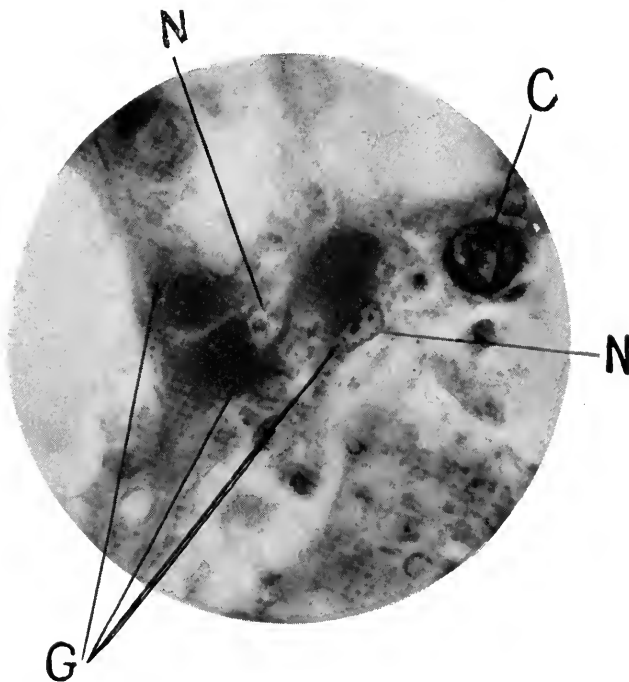


Fig. 2.—Three protoplasmic glia cells (*G*) with the nuclei (*N*, *N*) located at the very periphery. The processes of the homogeneous cell bodies are also quite distinct. At *C* is a capillary with pronounced endothelial cells and a shoe-shaped polymorphonuclear leukocyte. Alzheimer-Mann stain; $\times 800$.

Summary of Findings.—The findings were: imbecility, loss of spatial sense, marked paralysis of the third, fourth, fifth, sixth, seventh, ninth and twelfth nerves, with partial involvement of the tenth and eleventh nerves on both sides.

The diagnosis was: imbecility, poliencephalitis superior and inferior.

Necropsy Findings.—(Permission was given to remove the brain only.)

The pia was smooth, easily detachable from the brain and shiny, except the portion over both occipitoparietal regions which showed marked hemorrhages. The convolutions were of normal size, not flattened and the sulci of usual depth. The gray matter of the midbrain and medulla appeared unusually dark, almost black, sharply contrasting with the surrounding white substance. Foci of hem-

orrhages or softening were absent: the ventricles were not enlarged and the ependyma not proliferated.

Microscopic Examination.—Various portions of the cortex, midbrain, medulla and cerebellum, including the pia-arachnoid and both third nerves, were studied in celloidin, paraffin and frozen sections. The staining methods used were toluidin blue, thionin, Alzheimer-Mann, Bielschowsky, Herxheimer and combined Alzheimer-Mann-Bielschowsky. As the main pathologic changes were expected in the midbrain and medulla, these regions will be described in somewhat greater detail.

Photomicrograph 1, showing the condition of the tegmental portion of the pons, gives a fair idea of the principal changes encountered in the midbrain

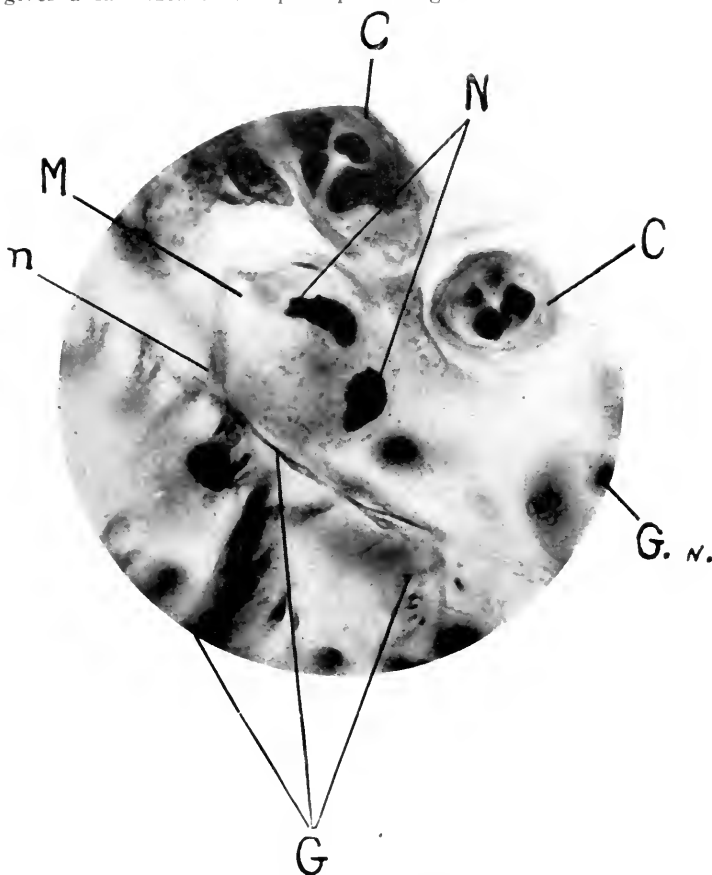


Fig. 3.—Tegmental region of the pons. *M* is a myelophage with a nucleus (*n*) and two fragments of an axon (*N*). *C, C* are capillaries containing four and five blood cells. The thick and other dark stained strips (*G*) are glia fibers; *G. n.* are glia nuclei. Bielschowsky stain; $\times 1200$.

and medulla. The visual field consisted of vacuolated glia tissue with wide meshes and a great amount of hyperemic and distended vessels. The smallest capillaries were unusually prominent and engorged with blood (Figs. 2 and 3). Often there could be found newly formed capillaries in the form of elongated

adventitial and hypertrophied, brightly stained endothelial cells. Such young vessels usually were free from blood elements. The adventitial spaces of larger as well as of smaller vessels, did not show infiltration cells, such as lymphocytes, plasma cells or polyblasts. Neither could polymorphonuclear cells be found, while in some instances erythrocytes could be seen scattered freely or enclosed within various gliogenous formations. In the large ganglions, especially the lenticular nucleus, the vascular walls sometimes appeared thickened and some-

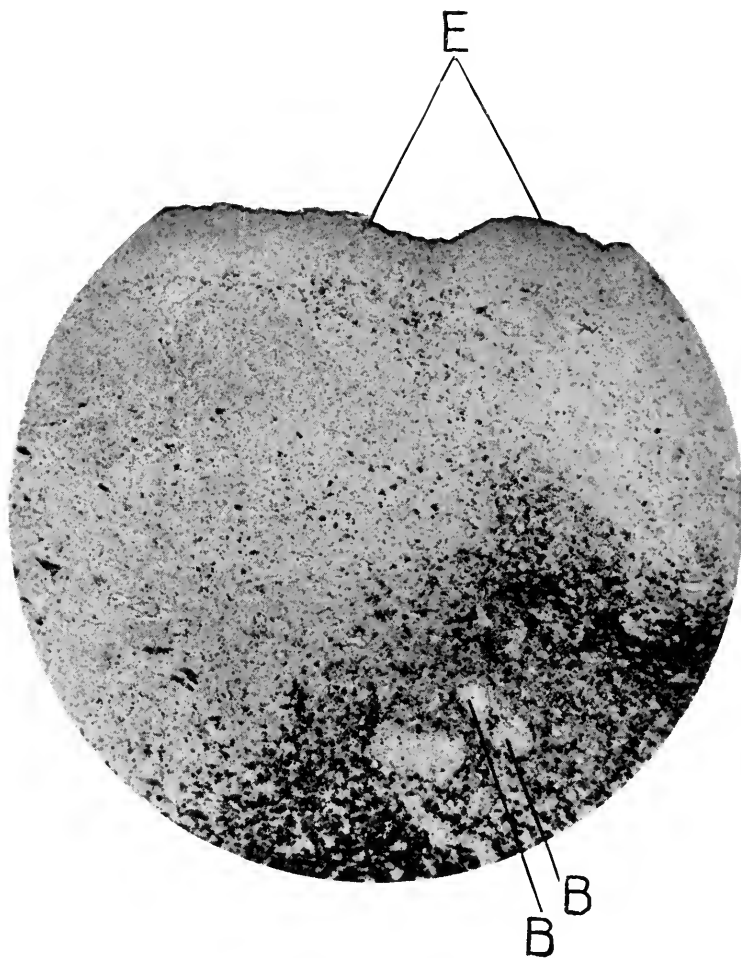


Fig. 4.—The region of the twelfth nucleus. The black masses are fat globules covering the area occupied by the twelfth nucleus and scattered as small droplets over the subependymal region. *B B* are blood vessels surrounded by fat enclosed within gitter cells; other pale areas in the darkened area are mostly vessels; at *E* is the ependyma of the fourth ventricle. Herxheimer scarlet-red-hematoxylin stain; $\times 60$.

times showed proliferation of adventitial and endothelial elements with an abundance of brownish-green, round granules. Similar vascular changes also

could be found in the cortical layers, especially in the occipital lobe, to a lesser extent in the cerebellum, and were quite pronounced in the choroid plexus.

The characteristic feature of the vascular changes was the almost universal presence of fat within or around the vessel walls, including those of the smallest capillaries. The latter, stained with scarlet red, showed enormous quanti-

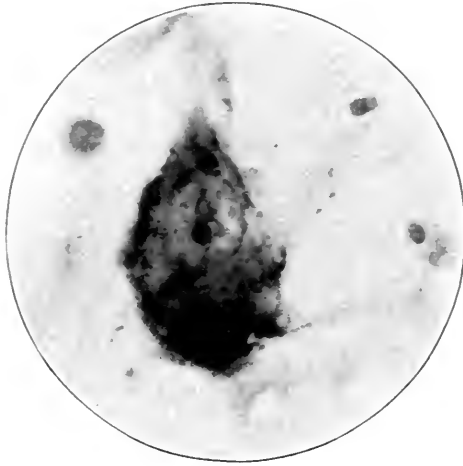


Fig. 5.—One of the types of ganglion cell changes; central chromatolysis and other changes. Toluidin-blue stain.

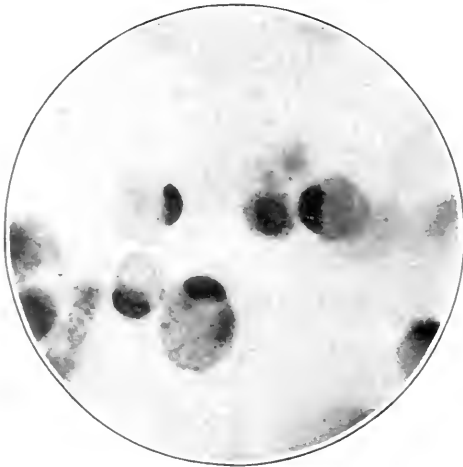


Fig. 6.—Gitter cells (7) of one of the degenerated areas. One cell contains two nuclei. Toluidin blue stain; $\times 1200$.

ties of fat which covered the entire visual field (Fig. 4), and made it impossible to discern any other structures except vessels and their contents.

Ganglion Cell Changes.—The ganglions sometimes appeared quite normal, even in those regions of the midbrain which exhibited marked structural

changes in the glia and blood vessels. Many nerve cells were decidedly pathologic, appearing atrophied, excavated and surrounded or invaded by a great number of glia cells. Other cells were sclerosed or homogeneous with deeply stained dendrites, dustlike chromatin and a dislocated nucleus poor in chromatin but possessing a well preserved membrane (Fig. 5). In short, chromatolysis, satellitosis and neuronophagia were the usual changes found in the ganglion cells. Stained with scarlet red, they always showed an abundance of fat drops and droplets over the entire cell body, including the nucleus.

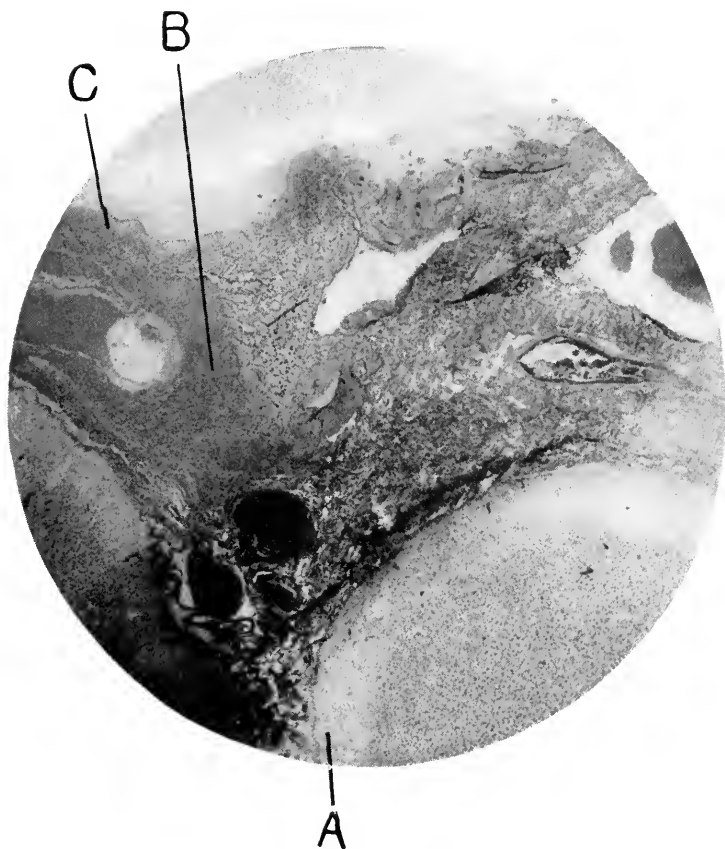


Fig. 7.—Cortex of the angular gyrus covered by a pia-arachnoid which is infiltrated with various cells and shows an abundance of blood vessels. The subpial layer *A* appears hyperplastic; *B*, hemorrhagic focus; *C*, arachnoid membrane. Toluidin-blue stain; $\times 16$.

The foregoing cellular changes, the fat-laden ganglion cells, together with the vast accumulation of fat around the extremely numerous vessels, were especially marked around the third and fourth ventricles and the Sylvian aqueduct; that is to say, they involved the nuclei of all cranial nerves, from the third to the twelfth, including the locus coeruleus, while the gray matter of the substantia nigra, nucleus ruber and basal portion of the pons were either entirely normal or showed but mild changes.

Glia Tissue Changes.—Marked as the changes in the gray matter were, those of the neuroglia were much more striking. The wide meshes of glia, appearing under the low power as empty vacuoles, exhibited, under the high power, especially with oil immersion, a great variety of unusual structures: (1) large

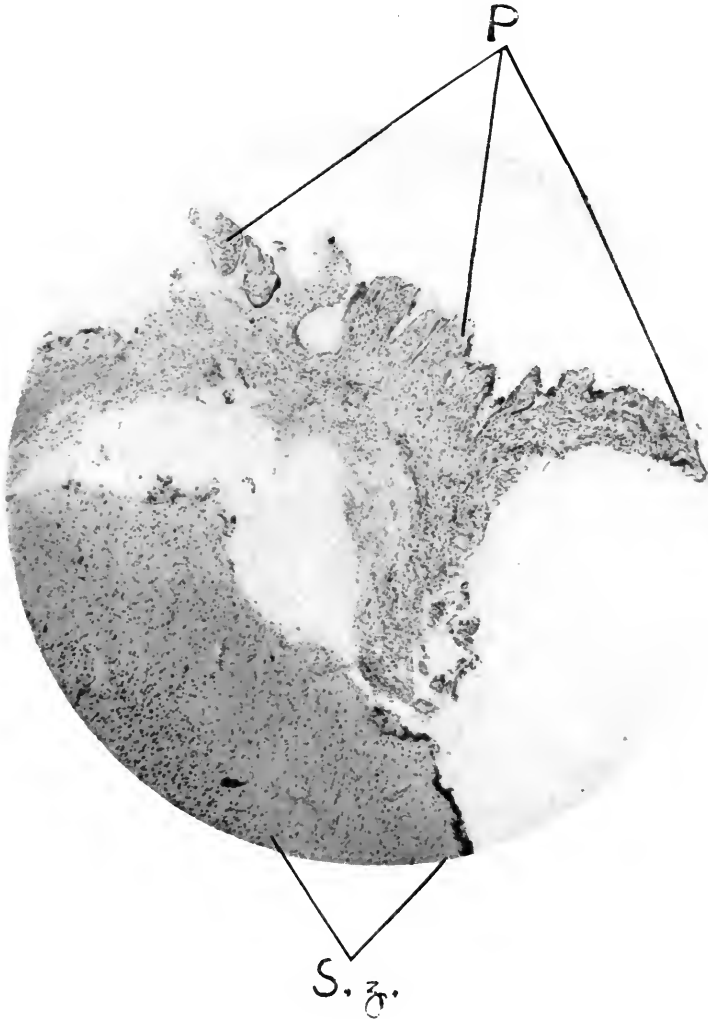


Fig. 8.—Pia-arachnoid of the motor area. The arachnoid is greatly infiltrated, mostly with glitter cells and mesothelial cells reproduced on photomicrograph 9. The arachnoid villi (pacchionian bodies) are well shown. *P* represents the pacchionian bodies (arachnoid villi); *S. z.*, the stratum zonale (subpial layer) is hyperplastic and covered by an abundance of glia nuclei. Toluidin-blue stain: $\times 36$.

round lattice-like reticular bodies, commonly described as "glitter" cells; (2) fragments of myelin and axons; (3) red blood corpuscles; and (4) various gliogenous formations (protoplasmic glia cells, myelophages).

The gitter cells were the most numerous elements, mostly in the form of round bodies wholly made up of fine vacuoles and without processes; their nucleus was flattened and misplaced to the periphery (Fig. 5). Some cells contained more than one nucleus and were abundant around capillaries and smaller vessels. Equally numerous were gitter cells that, in addition to the great mass of small vacuoles, exhibited one large vacuole containing remnants of broken-up nerve tissue. These are so-called gitter cells— α according to Jacob's classification.

Scarlet red stained specimens frequently exhibited a third variety of gitter cells, irregular in shape and possessing a great many processes packed with fat (gitter cells— β).

Aside from these three varieties of gitter cells, there were numerous other round formations with several large vacuoles, of which some were filled with

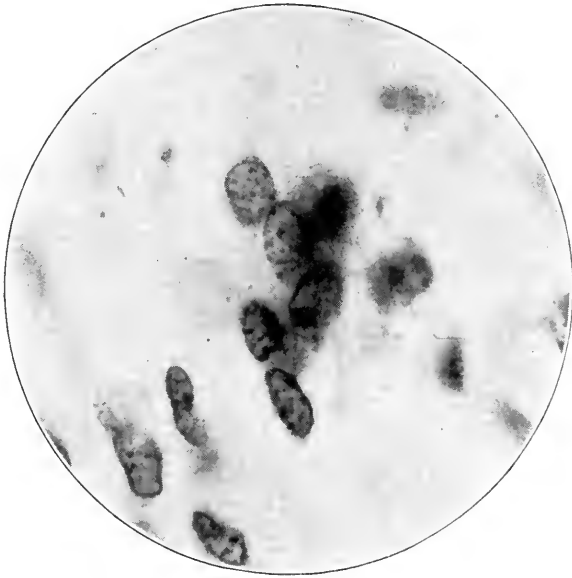


Fig. 9.—Clusters of mesothelial cells of the subarachnoid space. Toluidin-blue stain; $\times 1200$.

red blood cells and remnants of axons and myelin, well shown in photomicrograph 3. These are myelophages (Jacob) which, like the foregoing types of gitter cells, are gliogenous formations with a specific function. For instance, the function of the myelophages is to pick up the broken-up myelin or axon and transform it into fat which is stored up in the gitter cells. It is probable that the myelophages themselves become transformed into gitter cells after their contents, myelin and axon, have been, so to speak, digested and transformed into fat. The γ -variety represents the ripe stage of gitter cells and is to be found freely scattered or around the vessels to which they are supposed to deliver the fat for final elimination.

In the large ganglions (lenticular nucleus, optic thalamus, caudate nucleus) the basal portion of the pons, cerebral and cerebellar cortices, as well as in the

subarachnoid space only the γ -variety could be encountered, the other types including myelophages, being absent.

There were in the midbrain and medulla a great number of other atypical glia cells, so-called protoplasmic glia cells. Figure 2 shows such formations, rich in protoplasm, homogeneous in appearance and well supplied with processes. The chromatin-rich nucleus was misplaced toward the periphery of the cell body as if about to desert it. The processes, in many cells, were very large and stretched for a long distance, breaking up into numerous glia fibers. Often they enveloped red cells or remnants of broken-up brain tissue, such as ganglion cells, myelin globules and even smaller capillaries. The size of the proto-

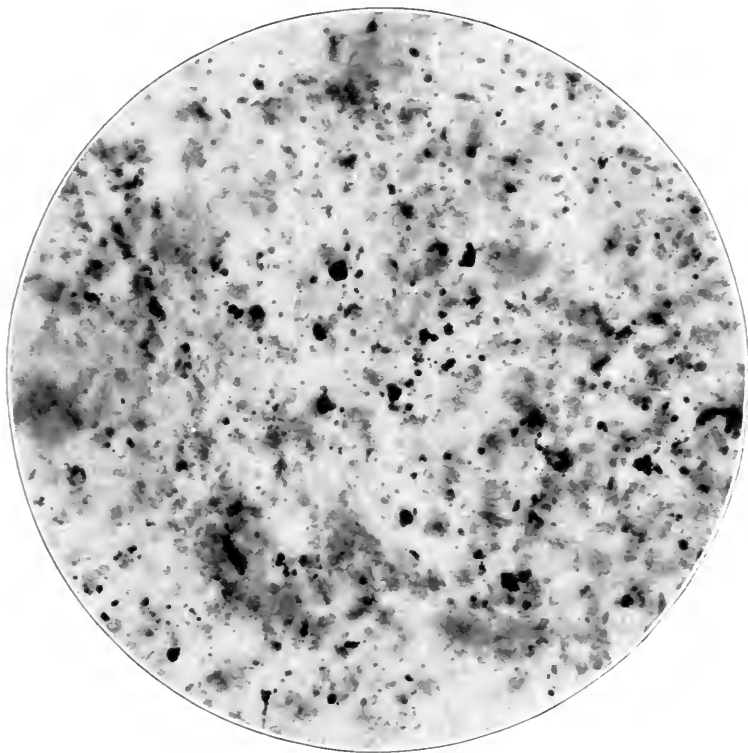


Fig. 10.—Fat globules (black dots) in the subarachnoid space. Herxheimer scarlet-red stain; $\times 230$.

plasmic glia cells may be quite large, somewhat resembling the monster or giant glia cells of a spinal cord involved in secondary degeneration.

Finally, many glia cells were represented by mere nuclei which, however, were always increased in size and rich in chromatin. This type of glia cells predominated in the larger ganglions and basal portion of the pons and cortex. Stained with scarlet red they showed minute globules of fat.

Nerve Fibers.—These were frequently pale, poorly stainable, sometimes swollen or tumefied, and therefore irregular in shape. Frequently they were broken up into smaller fragments enveloped by glia fibers; or they formed globules of myelin (Marchi globules) enclosed within the previously described

gliogenous formations. Frequently a changed nerve fiber was accompanied by, or made up of, strips of thin, delicate, densely red fibrils (degenerated axons).

The cortical layers, which were studied from every portion of the brain and cerebellum, showed changes analogous to those of the large ganglions, namely, excessive vascularization, numerous protoplasmic glia cells filled with fat droplets and various ganglion cell changes (neuronophagia, cell sclerosis and chromatolysis). Equally well represented all over the cortex, they were especially marked in the occipital lobe, the region of cuneus and precuneus and especially the angular gyrus.

The latter was studied with particular care, in view of the history of disturbances of the spatial sense which is supposed to be located in this portion of the cortex. Photomicrograph 7 shows this part of the cortex covered with

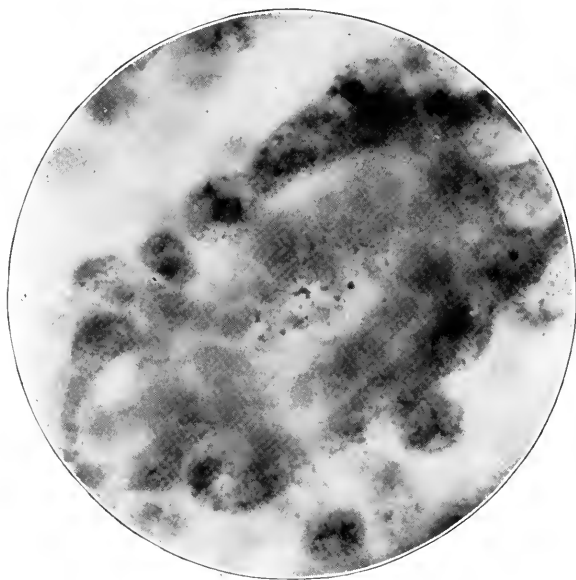


Fig. 11.—Choroid plexus. An engorged vessel surrounded by enlarged cystic epithelial cells showing granules within the cytoplasm. Toluidin-blue stain; $\times 800$.

an enormously distended and hemorrhagic pia-arachnoid which also exhibits unusually interesting findings over the rest of the cortex, especially on the base of the brain.

The distended meshes of the pia-arachnoid were packed with a great amount of various types of cells (Figs. 7 and 8). The latter were principally mesothelial (Fig. 9) and gitter cells mixed with an abundance of red cells. There were also in evidence numerous minute granules covering the meshes, the mesothelial (around their nuclei) and partly the gitter cells. Surface frozen sections of the pia arachnoid exhibited great masses of fat (Fig. 10) enclosed within gitter cells and gathered around the capillaries. The latter were numerous, distended, showed well stained adventitial and endothelial cells but no infiltration elements, such as lymphocytes, plasma cells, etc.

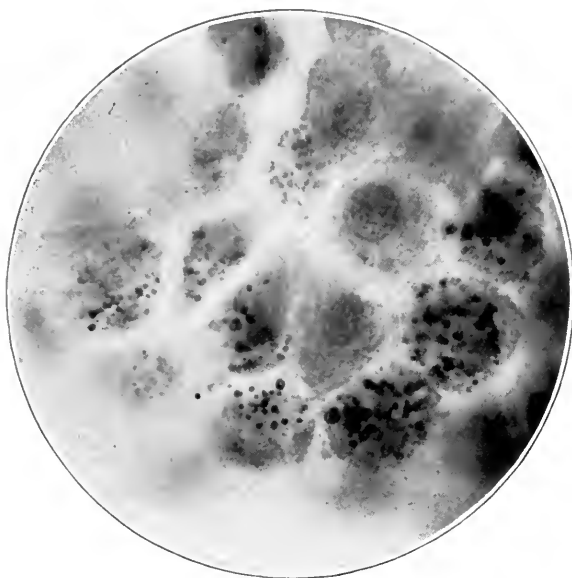


Fig. 12.—The epithelial cells of the choroid plexus packed with fat granules. Herxheimer scarlet-red; $\times 1200$.

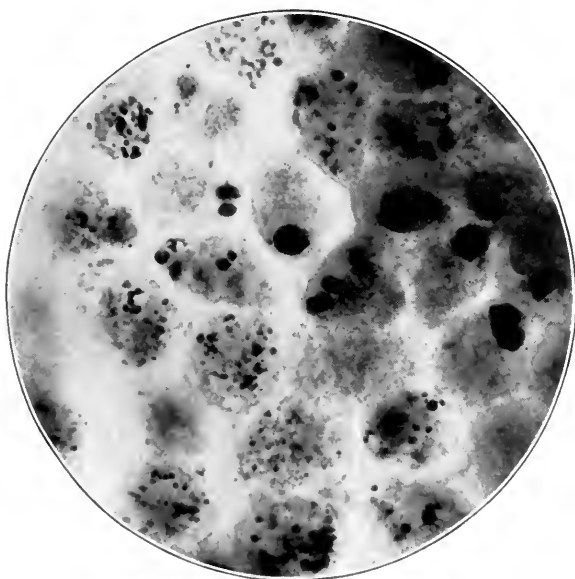


Fig. 13.—Same as photomicrograph 12. Some of the cells assumed the shape and form of gitter cells. Herxheimer scarlet-red stain; $\times 1200$.

In some toluidin blue specimens could be seen cell bodies with pale chromatin-poor nuclei surrounded by a rim of metachromatically stained cytoplasm. They were mostly irregular in shape and numerous in the arachnoid portion of the pia. Such ill-defined elements most probably were changed mesothelial cells which, like the typical cells, could be seen all over the subarachnoid space, regardless of the localization. They were quite numerous on the base, while over the angular gyrus they were obscured by extensive hemorrhages.

Noteworthy changes were also found in the choroid plexus. The vessels were engorged, the endothelial and adventitial cells of the capillaries promi-

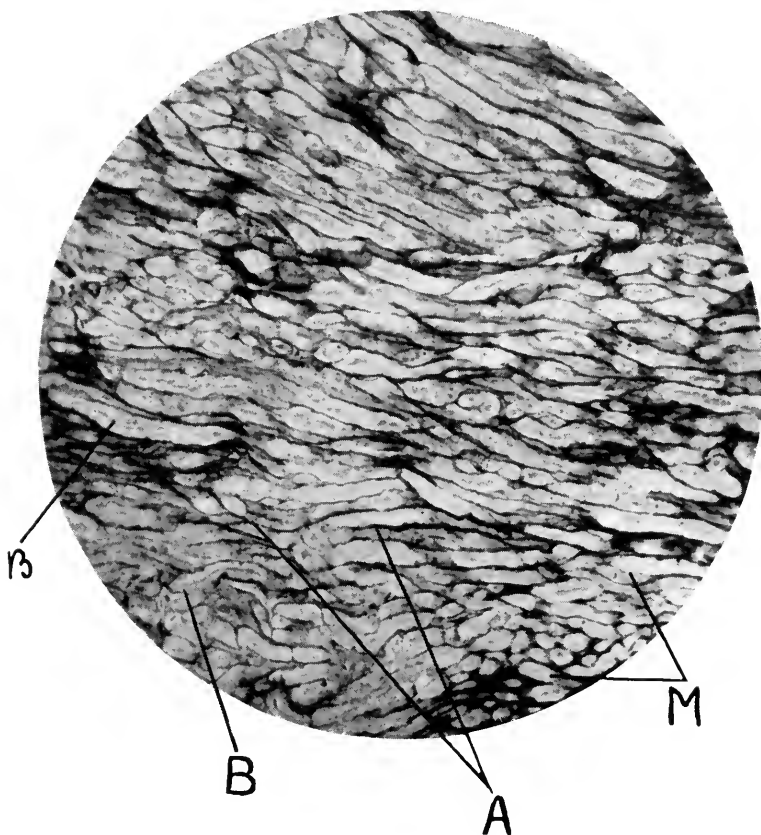


Fig. 14.—The third nerve broken up into numerous fragments; the axons can be well seen in the majority of the nerve fibers. The right lower area is filled with Marchi globules. *A* represents the Schwann membrane; *B B*, axons; *M*, Marchi globules. Alzheimer-Mann-Bielschowsky stain; $\times 150$.

nent and the epithelial cells surrounding the vessels appeared as unusually large, expanded, reticular bodies which were well stainable and contained a peripheral nucleus (Fig. 11). Unstained specimens exhibited the usual black granules, while those stained with scarlet red showed a great number of fat droplets scattered over the cytoplasm (Figs. 12 and 13).

The third nerve, in its distal portion, was converted into numerous so-called Marchi-globules (Fig. 14), that is to say, fragments of myelin and axons, enveloped by proliferated glia tissue (Schwann cells). These changes are well represented in Figure 14 and are characteristic of secondary nerve degeneration in its early stages.

The spinal cord changes could be studied only in the upper cervical portion, which showed mild vascular and parenchymatous changes. These were even less pronounced than those of the cortex or basal ganglions.

Summary of Findings.—The findings were: widespread degenerative changes of every portion of the cerebrum and cerebellum with particular involvement of the midbrain and medulla; marked secondary proliferative changes in the glia tissue and, to a lesser extent, in the vessels; fatty infiltration of the vessels of the brain, pia-arachnoid and choroid plexus.

DISCUSSION

Quite throughout, the changes were particularly striking in the mid-brain and medulla, where they exhibited a typical picture of various stages of so-called secondary degeneration, as described by A. Jacob in experimental animals. Jacob's ¹ findings, discussed by me ² at some length elsewhere, resemble the conditions to be found in amyotrophic lateral sclerosis, subacute combined cord degeneration, progressive bulbar paralysis and, to a large extent, in tabes and multiple sclerosis. The case under discussion differs from the foregoing by the principal, though not exclusive, involvement of the midbrain and medulla oblongata. In this respect it is almost analogous to another mesencephalic lesion—lethargic or epidemic encephalitis. The latter, however, is a purely inflammatory process, due to some infection, while the case under discussion is not inflammatory or infectious. It is most probably caused by some organic toxin, as is evidently the case in amyotrophic lateral sclerosis, subacute combined cord degeneration and similar conditions.

The term poliiencephalitis superior hemorrhagica applied to certain cases, including the present one, is therefore a misnomer, as already pointed out by Schroeder,³ among others. First of all, hemorrhages are not essential. They may be entirely absent (Oppenheim and Cassirer ⁴), or, as in this case, be so small that only careful microscopic search reveals them. It is therefore altogether illogical to look on these microscopic hemorrhages as the cause of the widespread degenerative changes

1. Jacob, A.: Ueber die feinere Histologie der secundären Faserdegeneration in der weissen Substanz des Rückenmarks, *Nissl-Alzheimer's Arb.* **5**:1, 1912.

2. Hassin, G. B.: Histopathological Changes in a Case of Amyotrophic Lateral Sclerosis, *Med. Rec.* **91**:228 (Feb. 10) 1917.

3. Schroeder, Paul: Zur Lehre von der akuten hämorrhagischen Poliiencephalitis superior (Wernicke), *Nissl-Alzheimer's Arb.* **2**:145, 1908.

4. Oppenheim, H., and Cassirer, R.: Die Encephalitis, in Nothnagel's *Specielle Pathologie und Therapie*, Wien. **9**: Pt. 2, 39, 1909.

and the marked progressive glia phenomena, an opinion expressed by Schroeder. The cause as stated is probably some organic toxin or catabolic phenomena which seems to possess the peculiar ability to affect certain portions of the central nervous system leaving others intact. For instance, the pure motor elements may alone be affected and give rise to the clinical syndromes of amyotrophic lateral sclerosis, bulbar paralysis or progressive muscular atrophy; or the white substance, principally of the spinal cord, may be exclusively involved, leaving the motor and sensory elements undamaged and thus produce a condition of subacute combined cord degeneration. The action of the problematical (catabolic) toxins is thus selective causing a great variety of morbid conditions which, though pathologically similar, are dissimilar in localization.

The histologic studies of this case bring forth some other interesting facts. The presence of fat granule bodies (gitter cells) in the subarachnoid space and the choroid plexus, as well as in the perivascular spaces, all over the brain stem and the cerebellum can be explained only by assuming that they reached these structures, or had been transported to them, from the degenerated areas of the midbrain. In other words, the great masses of fat were gradually drained from the midbrain regions by way of perivascular spaces toward the subarachnoid space.

The latter thus may be looked on as the receptacle of the waste products discharged by the brain tissues. Some of these waste products are lipid substances, some probably are represented by the numerous granules in the subarachnoid space. The proliferation of the mesothelial cells and the abundance of other, mostly ill-defined, elements is probably a reaction against the invading waste products, just as they react against invasion by some particular matter (Essick,⁵ Weed⁶).

When these products reach the subarachnoid space they are removed thence by the cerebrospinal fluid. If the latter is to be considered as the product of the choroid plexus, then the function of the cerebrospinal fluid is to wash off the waste products that come from the brain to the subarachnoid space.

The choroid plexus, however, exhibited an abundance of fat in the epithelial cells and its vessels. The fat could come only from the spinal fluid which thus gets rid, with the help of the choroid plexus, of some of its contents—in this case of the lipoids.

One gains an impression that the spinal fluid is entirely made up of the tissue fluids of the brain and is being continually discharged by way

5. Essick, C.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter, Publication 272 of the Carnegie Institution in Washington, pp. 377 and 388.

6. Weed, L. H.: The Cells of the Arachnoid, *Bull. Johns Hopkins Hosp.* **31**:343 (Oct.) 1920.

of the perivascular spaces of the cerebral vessels into the subarachnoid space. Weed⁷ admits such a possibility though he believes that only small amounts of cerebrospinal fluid originate in this manner. Yet, coming as it does from such a voluminous and active organ as the brain, the cerebral fluid necessarily must be abundant and rich in proteins and various waste products. Before being absorbed by the arachnoid villi and other channels, the fluid becomes cleared, as it were, by the choroid plexus and thus rendered more passable.

Further pathologic studies of cases like the one recorded will, to a great extent, facilitate the solving of these highly difficult and interesting problems. The facts brought forth by histopathologic studies of the subarachnoid space and the choroid plexus may show that the function of the cerebrospinal fluid and the choroid plexus is altogether different from the one generally accepted.

CONCLUSION

1. Poliencephalitis superior (hemorrhagica) of Wernicke is not an inflammatory, but a partial manifestation of a general degenerative process of the central nervous system.

2. It is analogous to other degenerative processes, such as amyotrophic lateral sclerosis, subacute combined cord degeneration, progressive bulbar paralysis and tabes dorsalis, from which it differs by the localization of the degenerative phenomena.

3. It is essentially, though not exclusively, a mesencephalic lesion, resembling in its localization epidemic encephalitis which, however, is not a degenerative but an inflammatory process.

4. The subarachnoid space is a receptacle of the tissue fluids which carry away the waste products of the brain.

5. The function of the choroid plexus is probably to pick up from the cerebrospinal fluid harmful or other products and to render them, as well as the fluid, more absorbable.

7. Weed, L. H.: Studies on Cerebrospinal Fluid, Article IV: The Dual Source of Cerebrospinal Fluid, *Jour. Med. Research* **31**:93, 1914.

THE MONGOLIAN IDIOT

A PRELIMINARY NOTE ON THE SELLA TURCICA FINDINGS

WALTER TIMME, M.D.

NEW YORK

In the past year it has been my great privilege to examine a number of Mongolian defectives at Waverley, Mass., at the invitation of Dr. Walter E. Fernald. The case reports, complete clinical data, biochemical findings and the result of treatment on an endocrinologic basis will be the substance of a later communication by Dr. Fernald and myself. I desire to make at this time a preliminary publication of the findings of the roentgenologic examination of many of the skulls for the reason that it may stimulate the making of such examinations in many centers with possible corroboration of my findings. Furthermore, if necropsy material is available, a special effort might be made, on the basis of the sellar changes shown in the radiographs, to determine the pathology of the pituitary body.

In twenty-three out of twenty-four nonselected cases of Mongolian idiocy, including six of my own, the radiograph of the skull showed a peculiar change from the normal in the anterior portion of the fossa pituitaria. This change consisted in an excavation under the anterior clinoid processes and presumably under the olivary process and optic groove, and the excavation communicated directly with the anterior portion of the fossa itself. There were varying degrees of this excavation, as seen in the accompanying illustrations. From our knowledge, more or less exact, of the influence of the anterior lobe of the pituitary on growth and genital development, this roentgen-ray finding is of considerable interest. Especially is this true when we remember that among the clinical signs of Mongolian idiots we invariably have the combination of subnormal and disproportionate body growth coupled with lack of genital development. Thus in boys, undescended testicles are quite frequent and in girls abnormalities of the genitals with tardy menstrual flow or even complete amenorrhea are the rule. The intimate relationship which in early life exists between the anterior hypophysial lobe and the pharyngeal glandular elements is also strikingly coincidental with the extreme pharyngeal mucous secretion seen in Mongolian idiots. Furthermore, with such an excavation, involving at times the optic groove, eye symptoms should be of frequent occurrence. These

ocular manifestations have been the subject of much discussion among ophthalmologists and a rather complete résumé of their findings was published some years ago by Charles A. Oliver.¹ Among the findings are:

"The optic discs in a number of cases are unequally grayed, especially in the deeper layers and to the temporal sides; the substance of the disc in the great majority of cases, especially where the retinal and choroidal disturbance was not pronounced, is apparently edematous; the retina surrounding the optic nerve head is edematous and swollen in many of the cases; examination for visual field disturbances could never be properly carried out through lack of cooperation."

Theoretically, therefore, disturbance of the anterior portion of the pituitary body might readily produce many of the symptoms shown clinically by Mongolian idiots. It is therefore suggested that every necropsy examination in cases of Mongolian idiocy should include a careful examination of the pituitary gland, notably in its anterior portion. Should such an examination eventuate in a corroboration of antemortem findings, perhaps a rational treatment might be forthcoming for these cases. Indeed, even on the theoretical basis of anterior lobe disturbance, the writer has inaugurated a therapy in several of his cases which has had some degree of success thus far. In one of his Mongolian idiots the testicles have descended since treatment was begun, and there seems to be a measure of mental improvement likewise. This treatment includes the hypodermatic injections of anterior lobe extract (antuitrin) combined with whole gland feeding and thyroid administration in small doses.

It is important to observe, nevertheless, that excavation under the anterior clinoids and optic groove is seen occasionally in persons who have not Mongolian characteristics, but who have isolated symptoms and signs due to anterior lobe disturbance, namely, growth and genital developmental abnormalities.

Speculation as to the character of the contents of this excavation would at the present be idle. Suffice it to say, however, that on theoretical grounds it should be tissue foreign to the normal anterior lobe type and possibly interfering with whatever of normal structure may remain in the region.

There is included in the series of photographs accompanying this communication one of a Chinese girl, 13 years of age, who is normal in all ways, in order that a comparison may be made of her sella turcica

1. Oliver, C. A.: *Med. Rec.*, Oct. 3, 1891.

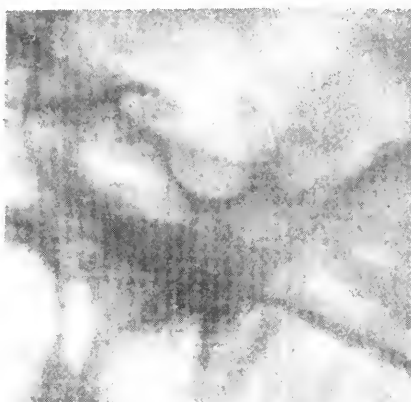


FIG. 1



FIG. 4



FIG. 2



FIG. 5



FIG. 3

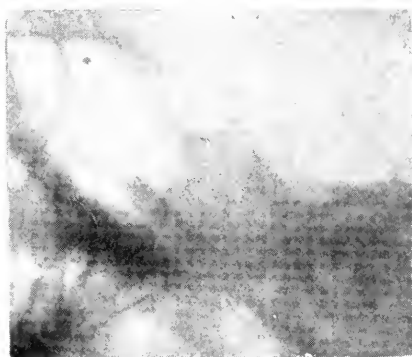


FIG. 6

Fig. 1 (T. J., girl, Waverley).—The sella turcica is rather generally enlarged with a marked excavation anteriorly opening directly into the general pituitary fossa.

Fig. 2 (S. H., girl, author's patient).—Anterior excavation prominent and even somewhat larger than the pituitary fossa itself.

Fig. 3 (T. S., boy, Waverley).—Similar to Figure 2.

Fig. 4 (L. H., girl, author's patient).—Anterior excavation rather different from foregoing in that while it is fairly long, its width is less.

Fig. 5 (E. M., girl, Waverley).—Similar to Figure 4, with a narrow and rather short cavity anteriorly.

Fig. 6 (Chinese girls).—Normal sella turcica. Kindness of Percy Ashley.

with that of the Mongolian idiot. It will be seen that in her skull the pituitary fossa seems to be normal in all particulars, however different the other skull landmarks may appear to be.

From time to time observers have been adding additional findings in their reports on Mongolian idiocy, but these findings have been practically all in the symptomatologic domain. The present report of

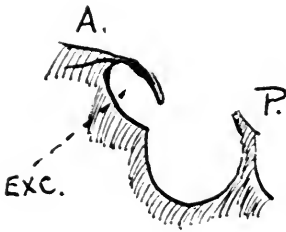


FIG. 1

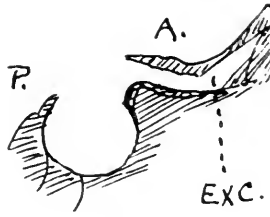


FIG. 4

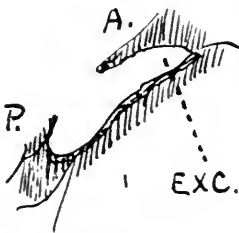


FIG. 2

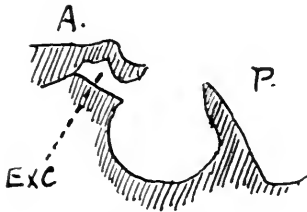


FIG. 5

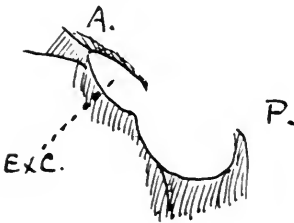


FIG. 3

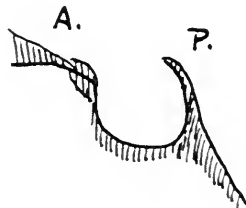


FIG. 6

Fig. 7.—Diagrams illustrating sella photographs. A, anterior clinoid process, P, posterior clinoid process; Exc., excavation.

the sellar changes, if substantiated by the necropsy examination, is of quite a different nature, for it furnishes at once a basis for the structural changes of the Mongolian idiot and for the greater part of his symptomatology, and hence must be regarded as one of the fundamentals of the situation. Furthermore, it offers an angle of attack in therapy.

SYPHILIS OF THE EIGHTH NERVE *

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In view of the fact that there is no affection of any cranial or spinal nerve that is more characteristic or more easily recognizable, and scarcely any other, unless it be invasion of the optic nerves, that is followed by more lamentable results, it is rather remarkable that syphilitic disease of the eighth nerve had apparently attracted but little notice among American and English neurologists until comparatively recent years. This was so in spite of the fact that Jonathan Hutchinson had called attention to it, and had even made it one of the triad of symptoms in hereditary syphilis. He had also referred to absolute deafness coming on rapidly in secondary syphilis, usually bilateral; but he had given no systematic description of the symptoms that distinguish the affection of the cochlear nerve from that of the vestibular. Gowers has an inadequate reference to the subject; he speaks of primary degeneration of the eighth nerve in locomotor ataxia, but he has little to say about acute syphilis of the acoustic nerve. The older American textbooks have only passing allusions to the subject. Mills gives a more complete, but condensed, account. He refers to the theory of Althaus that the vestibular suffers more than the cochlear nerve in tabes, due to a degenerative process, causing vertigo.

It is to the otologists that we must look for more complete work, because the subject is generally assigned to them, and there is a large bibliography. West, in Power and Murphy's "System," gives a good description of syphilis of the internal ear and auditory nerve. He seems to think that the early sudden cases are due to labyrinthitis, and the late tertiary cases to meningitis and neuritis. He gives a long bibliography. Rosenstein, in 1905, studied the changes in the auditory nerve, and found basilar gummatous meningitis. In Hazen's recent work on "Syphilis" the chapter on the ear is written by Dabney, who reviews the subject and gives many references, mostly to the otologists who have written on it. Fournier, in his work on "Syphilis," has a short chapter written by Hermet, who speaks of the rapidity and incurability of syphilis of the eighth nerve, some cases occurring as early as the fifth and sixth months after the primary infection, sometimes accompanied with paralysis of the seventh nerve. The best recent review of the subject in English, of which I have knowledge, is by Dr. G. W. Mackenzie of Philadelphia, in a paper on "Syphilis of the Inner Ear and

* From the wards and laboratory of the Philadelphia General Hospital.

Eighth Nerve." I must leave to the otologists their own field, for I am writing as a neurologist and largely, but not entirely, from a clinical standpoint.

My attention has been called anew to this subject by the recent occurrence of early and striking cases in hospital practice. This may be due in part to the intensive study of syphilis of the nervous system which has followed upon our knowledge of the spirochete and our advanced laboratory methods. We have come to know how early the spirochete may invade the central nervous system, and we are naturally on the lookout for any and all manifestations of it. Cases of deafness, tinnitus and vertigo, which formerly would probably have been promptly sent to the otologist, are now retained by the neurologist and scrutinized most closely. This is proper and necessary, because few, if any, of these cases are strictly otologic. They are primarily nervous cases; the lesion is in the nervous system; and the involvement of the eighth nerve is nearly always associated with other well marked symptoms of nervous syphilis.

CHARACTERISTICS OF SYPHILIS OF THE EIGHTH NERVE

There are certain characteristics of syphilis of the eighth nerve which the neurologist should bear in mind, particularly as he is quite as likely as the otologist or syphilologist to see these cases in their earliest stage.

First, the onset may occur early in the secondary stage, sometimes very early; and even before the secondary stage, if we are to believe Pollitzer, who claimed that he saw a case seven days after infection. Randall saw a case in which deafness occurred four weeks after an infected needle-wound of the finger. Other writers speak of its early appearance, and this has been true in my own observation, the disorder showing itself in a few months.

Second, suddenness of onset and rapidity of course are sometimes striking. The patient may become deaf in a few days. Hermet spoke of "*surdité foudroyant*." Dabney says that "sudden loss of hearing, generally with tinnitus, no pain, no evidence of middle-ear disease, in a young adult otherwise healthy, should be regarded as almost certainly indicative of syphilitic disease of the eighth nerve or labyrinth." This tinnitus is usually marked and most distressing, even keeping the patient awake at night. It is, of course, evidence of involvement of the cochlear nerve.

Third, the disease is usually bilateral, seldom unilateral, as Hutchinson pointed out.

Fourth, there may be a cranial polyneuritis, the seventh nerve especially being paralyzed with the eighth. Sometimes the second, third, and fifth, one or all, are involved. Nonne and others have called

attention to this fact as evidence that the affection is primarily a basilar meningitis, not a labyrinthitis as some of the older otologists taught. As evidence that we are dealing with a basilar syphilitic meningitis is the fact that severe headache, with high lymphocytosis of the spinal fluid, may precede or accompany the neuritis. But this apparently is not so in all cases.

Fifth, the two divisions of the eighth nerve may not be equally involved. The cochlear in one case, the vestibular in another, may be the more affected. This is in accord with the well-known selective action of syphilis. The vestibular nerve should always be tested by the Bárány methods.

Finally, the disease may be incurable, causing complete deafness in a short time.

THE COCHLEAR AND VESTIBULAR NERVES, THE SO-CALLED ROOTS OF THE EIGHTH NERVE

It is to be borne in mind that the eighth nerve, although usually described by anatomists as one nerve with two branches, is really two distinct nerves, each with its own ganglion of origin, its own nerve trunk, and its own separate and distinct course and distribution in the central nervous system. For a part of their course these two nerve-trunks are bound together (in the internal auditory meatus) so that they appear as one nerve, but before entering the brain-stem they separate, and form the so-called roots of the eighth nerve—but they are not roots in any true sense, because, like all sensory neurons, these have their ganglions of origin outside of the central nervous system. The cochlear nerve arises in the ganglion spirale, or ganglion of Corti, within the labyrinth; the vestibular, in the ganglion of Scarpa, which is located within the internal auditory meatus. The fact that these two so-called roots are distinct before entering the brain-stem, explains how they can be separately affected in syphilitic meningitis. The fact that the eighth nerve has no neurilemma (hence called the *portio mollis*) may explain its vulnerability to the spirochete, and the extreme rapidity of the destructive action.

Disease of the cochlear nerve causes deafness of various grades, which is to be determined by tests made by the otologists. It has been claimed by some that abolition of the very high and very low notes, with preservation of the intermediate notes, hence allowing the hearing of a conversational tone, is quite characteristic. This was present in one of my cases; but Mackenzie denies that it is common, and says that various notes are abolished in various cases. Appropriate tests are likely to show impairment of bone conduction. It is necessary to exclude disease of the middle ear.

The vestibular nerve presides over equilibration, and its central connections are with nuclei in the brain-stem, from which by a second relay of neurons its ultimate connections are made, especially with the cerebellum and the nuclei in the midbrain. Vertigo and nystagmus, which are normal reactions on turning and douching, are variously affected, as can usually be demonstrated by the Bárány tests. Clinically, these patients may have various forms or degrees of vertigo, but in my observation staggering gait is not common.

I do not attempt here to describe tests and reactions in detail, for this province belongs to the otologist, whose cooperation is always essential in a proper study of these cases. I believe, however, it is necessary to be on guard against hasty conclusions as to the localizing value of the Bárány tests. In my observation these tests in these cases are mainly valuable in showing alteration or complete abolition of the normal vertigo and nystagmus on turning, the inference being that the nerve trunk is implicated.

It is not to be denied that there may be a true syphilitic labyrinthitis. Some otologists seem to have held that this was the common pathology, especially in the early acute cases. It may be so also in the hereditary cases, in which the bone also may be diseased. The distinction between disease of the labyrinth and disease of the nerve is, I understand, not an easy one, and it is a problem for the ear specialist rather than for the neurologist; but there is little doubt, for reasons which I have already given, that the meninges and nerve trunk are the seats of the disease in many cases, if not in the majority.

The following case may serve as an example of the fulminating type of this disease:

REPORT OF CASES

CASE 1.—A negro, 30 years old, had been treated in the venereal wards of the Philadelphia General Hospital in June, 1917, for a chancre and secondary eruption. At this time he had received one dose of arsphenamin. He had later a syphilitic iritis, but there is no record of any nervous symptoms at this time. He left the hospital and returned to his work, but about two months later he was taken with a severe frontal headache, which forced him to return to the hospital. The headache was the only complaint. The right pupil was rigid, the result of the iritis; the left was sluggish. The cranial nerves were normal. The knee and Achilles' reflexes were much diminished, but there was no ataxia or swaying. As a routine measure the hearing was tested; no deafness was observed. Both the blood and cerebrospinal fluid were strongly positive, and there was a very high lymphocytosis. He was given mercurial inunctions and several doses of arsphenamin. In October the report from the ear clinic stated that hearing was normal; late in November, however, the hearing was slightly impaired, but the drum membranes were normal. Under treatment the headache entirely disappeared, and the blood became negative; the spinal fluid remained positive, though the cell count was much reduced. Toward March the man eloped from the hospital and was gone several weeks, when he returned. In

that short interval he had become very deaf, to such a degree that it was difficult to speak with him. He complained much of loud tinnitus. The report from the ear clinic stated that in the right ear the involvement seemed to be confined to the labyrinth, but in the left ear the indications were that the lesion was in the course of the nerve. Bárány tests were not made, but the patient had no vertigo or staggering. Active treatment did not relieve the deafness, which indeed increased until in a short time it was practically complete. There was a smoothing out of the face and brow on the left side, due to slight paresis of the seventh nerve.

The Bárány tests may give interesting results in some of these cases, as the following instance shows. It also shows bilateral seventh nerve palsy, which in itself is a rarity.

CASE 2.—The patient, a colored man, aged 23, had a primary sore in September, 1918. He was admitted in October to the venereal wards of the Philadelphia General Hospital, where he was given two doses of arsphenamin; he left the hospital against advice on December 1. Three weeks later (or about three months after the appearance of the chancre) he began to have severe and persistent headache, accompanied with vertigo and tinnitus in both ears. He was readmitted to the hospital in January, when it was observed that he had paralysis of both facial nerves, more marked on the left. Other cranial nerves were not involved (with the exception to be noted) nor was there any impairment of the spinal cord, but the Achilles' reflex on the left side was abolished. The pupillary responses were normal; there was no optic neuritis. The Wassermann tests of the blood and spinal fluid were positive, and the cell count was as high as 780. He received six doses of arsphenamin up to March, 1919. The positive but shortened Rinne's test, with loss of intermediate notes, pointed strongly to involvement of the nervous mechanism; but the patient heard ordinary conversation. Dr. Lewis Fisher reported total absence of response after douching and turning, which would indicate a lesion of the eighth nerve (vestibular division); but a definite presence of vertigo after turning, as well as a preservation of a fair amount of hearing on both sides, showed that the eighth nerves were not involved in their entirety. Dr. Fisher suggested a bilateral lesion of the brain-stem on the mesial aspect of Deiters' nucleus, thus allowing the escape of the fibers for vertigo and most of the auditory fibers; but the involvement of both seventh nerves was against this view, for it clearly indicated a peripheral lesion of the so-called roots of the eighth nerves, especially the vestibular, along with the seventh nerves, in a syphilitic meningitis. As already said, it is quite conceivable that these two roots may not be equally involved in a meningitis. It is noteworthy that this patient's seventh and eighth nerve involvement showed itself three months after the primary lesion, and after he had received two doses of the arsenical drug.

The following case is of especial importance because it is the only case in the series in which it was possible to examine the eighth nerve under the microscope. It is also of much clinical interest. As in the two preceding cases, the patient was under observation from the time of the initial lesion.

CASE 3.—The patient, a white man, aged 48, was admitted to the venereal wards in September, 1918, with a primary sore. He received five doses of arsphenamin. In March, 1919 (six months after the primary sore), he began

to have a staggering gait, which caused him to fall to the left. Trouble with hearing had commenced earlier in the right ear, in which he had become quite deaf; later the left ear failed. There was loud tinnitus. The blood Wassermann reaction was reported negative, but the spinal fluid was + + + +. The cell count was 910. There was complete abolition of the left Achilles' jerk, just as in the other case (an odd coincidence). A low-grade double optic neuritis was present, but the pupils acted normally. The frontalis muscle on the left was smoothed out. All the other cranial nerves were normal. Dr. Fisher found that the right labyrinth was completely, the left partly, involved. There were no reactions to turning or douching. The case seemed to be a clear one of peripheral involvement, that is, of the labyrinths and eighth nerves in both divisions. The earliest symptoms appeared in less than six months after the primary sore. This man was taken with acute appendicitis and died. Under the microscope the eighth nerves were seen to have been affected, as shown in the following report.

Neuropathologic Report (by Dr. N. W. Winkleman).—Pathologic study was made of sections from representative areas of the cerebrum, cerebellum, pons, medulla, spinal cord and the eighth nerves. The stains used in this study were: a hematoxylin-eosin, Weigert's myelin sheath stain, Mallory's phosphotungstic-acid-hematoxylin and Alzheimer-Mann.

The cerebrum and cerebellum are normal, especially in so far as syphilis is concerned. The pons is negative. In the medulla there is seen a round cell infiltration (lymphocytes) in the nucleus vestibularis lateralis (Deiters') with fatty degeneration of the nerve cells of this nucleus on the right side. The membranes are normal. The spinal cord is negative.

Both eighth nerves show some swelling and tortuosity of the axis cylinders, as seen in longitudinal section, with some dropping out of axis cylinders as seen in cross section. There are present within the nerves large, round, vesicular, pale staining nuclei with irregular, indefinite, pale, acid-staining cytoplasm—in all resembling glia cells, and in places fibers are seen coming off the cytoplasm. Besides these there is present another type of cell: a long, narrow, heavier staining nucleus with a definite outline to its rather meager cytoplasm—a so-called sheath cell. At certain places within the nerves is seen a slight but definite lymphocytic infiltration. Amyloid bodies are present to excess. Around some of the vessels within the nerves are a few plasma cells with many lymphocytes. The sheath of the nerves shows a very definite though not very heavy infiltration with lymphocytes.

Diagnosis: Syphilis of both eighth nerves with involvement of Deiters' nucleus on the right.

The following case, which occurred in the service of Dr. Charles S. Potts, showed improvement under treatment with neo-arsphenamin (five doses) and mercurial inunctions. It was a late tertiary case.

CASE 4.—A white man, aged 56, began, one month before admission to the hospital, to have deafness, with vertigo and tinnitus in both ears. He also had impaired sight in the right eye and slight paresis of the right seventh nerve. There was a history of a primary sore fourteen years before. The left pupil was sluggish and irregular, the right inactive to light. Sight was much impaired. The right optic disk was much congested, with blurred margins. The Bárány tests revealed horizontal nystagmus to the left, four seconds; to

the right, four seconds. The tests of the blood and spinal fluid were positive on two examinations, but the cell count was low. Improvement was rapid under treatment. There was return of function of the vestibular, as well as of the cochlear, nerve. When discharged, the man could hear and see fairly well. It is noteworthy that this was a tertiary case of long standing, and that there was no lymphocytosis. This probably puts it in a separate class from the acute cases, already described, coming on in the early secondary stage, with headache and high lymphocytosis, indicating a rapid and acute involvement of the meninges.

CASE 5.—Another patient in Dr. Potts' wards was a negro, aged 28, who had become deaf rapidly, and who also had paralysis of the right seventh nerve. The pupils were irregular and sluggish. The clinical notes are not very full, as it was almost impossible to communicate with the patient, and he soon began to show mental symptoms, for which he was transferred to the department for the insane. No history of a primary sore could be elicited; the laboratory reports for blood and spinal fluid were negative, but there was a high lymphocytosis. He may have had a syphilitic psychosis. The onset of rapid and complete deafness in a young adult, with tinnitus, without obvious cause, and without disease of the middle ear is, as Dabney pointed out, significant of syphilis; and I may add that this is especially true if there is also a paralysis of the seventh nerve.

CASE 6.—This patient's condition was of five years' duration. He was a white man, 31 years old. He had bilateral seventh and bilateral eighth nerve paralysis and anesthesia on the right side of his face. The blood reaction had been reported several times as strongly positive. He had come and gone to and from the hospital four or five times; consequently the course of symptoms had not been accurately traced. His condition was evidently incurable, and illustrated the lamentable fate of a patient with this syphilitic syndrome. He was almost totally deaf, with paralysis and contractures of both sides of his face.

EFFECTS OF ARSPHENAMIN ON SYPHILIS OF THE EIGHTH NERVE

In discussing this affection, the subject of neurorecidivus, or the spinal fluid changes in neurosyphilis caused by a provocative dose of arsphenamin, inevitably occurs to the mind. The first three of the foregoing series of cases suggest this possibility. Another thing to be considered is the possible injurious action of the drug itself directly on the nerves. This charge has been made against this arsenical preparation not only in the case of the eighth nerves, but also in the case of the optic nerves. When, however, it is considered what a large number of injections of arsphenamin are being given every day, and what a comparatively small number of such complications occur, the inference that the drug is the cause is hardly warranted. Mackenzie criticizes the statement, made by some observers, that this affection of the eighth nerve has been caused by arsphenamin, and combats it. He believes that such cases are instances in which the treatment has not been sufficiently active. In the discussion of a paper by Klauder on this provocative action of arsphenamin, read before the Philadelphia

Neurological Society recently, Solomon said that Crockett, of the Massachusetts Eye and Ear Infirmary, had made a study of syphilitic nerve deafness before the arsphenamin era and after, and found the incidence about the same, if anything a little less, since the use of this drug. The absence of the Achilles' reflex on one side in two of the cases is noteworthy in this connection. Beeson¹ in a recent paper refers to abolition of the Achilles' reflex as a danger signal in treatment with arsphenamin. It may indicate a peripheral neuritis due to arsenic.

Under the microscope the appearance of the tissue is distinctly that of syphilis, as shown by the lymphocyte infiltration; but there is also the appearance of alteration of the nerve-fibers which is at least suggestive of the action of a poison.

1. Beeson, B. B.: Polyneuritis Plus Dermatitis Exfoliativa Following Neo-Arsphenamin, *Arch. Dermat. & Syph.* **2**:337 (Sept.) 1920.

LATE RESULTS IN EPIDEMIC ENCEPHALITIS *

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There were about 145 cases of epidemic encephalitis in the neurological and medical services at the Mount Sinai Hospital during the epidemics of this disease in 1919 and 1920. A great many of these patients left the hospital soon after the acute infection had subsided, showing some residual symptoms and signs of the disease. After a number of months had elapsed, we felt that it would be of interest to reexamine these patients in order to determine what, if any, changes were still present. With this object in view, this follow-up study was undertaken. Altogether eighty-nine patients were reexamined, and these examinations comprise the basis for this report.

In order to be reasonably certain that none of the symptoms or residual signs found at this reexamination were still part of the acute stage of the disease, at least six months were allowed to elapse before a patient was reexamined. The earliest cases were seen late in 1918, so that some of the patients were examined approximately two years after their acute illness. A complete neurologic examination was made in each case. No attempt will be made in this communication to correlate the symptoms and physical findings in the reexamination with those noted during the acute stage of the illness.

The findings in the cases may be grouped under these headings: (1) psychic disturbances, (2) tremor and irregular involuntary movements, (3) disturbances in attitude and gait, (4) disturbances in tonus and reflexes, (5) residual symptoms in cranial nerves, (6) pupillary disturbances, (7) epilepsy, (8) other residual symptoms and signs, (9) progressive cases, and (10) mortality in these 145 cases.

PSYCHIC DISTURBANCES

Fifty-one of the eighty-nine patients studied showed derangement in their psychic functions in one form or another; twenty-six of these complained of various degrees of irritability; six patients had become so irritable since their illness that they quarreled with everybody who came in contact with them. A number stated that they had no desire

* From the Neurological Service of the Mount Sinai Hospital, New York City.

* Read at the first annual meeting of the Association for Research in Nervous and Mental Diseases, Dec. 29, 1920.

to associate with any one, and preferred to be alone. They complained of restlessness, were easily disturbed, and would be startled and frightened at the slightest noise. They were unable to concentrate on any subject, and lacked interest in current events. Their mental reactions were retarded, and they were slow in following topics which were being discussed. Slight efforts produced undue physical and mental fatigue. These symptoms were particularly striking in those patients who showed symptoms pointing to involvement of the pituitary or basal ganglions.

Emotional instability was present in many of these patients, and in the majority it was evidenced in a marked depression. They were easily moved to tears, and worried continually—some because they could not sleep, and others because their various symptoms persisted so long after their acute illness was over. A number were worried because they still suffered from drowsiness. A few felt happy and unconcerned; they displayed a feeling of well being that was not in accord with what their physical examination disclosed. Two of these patients were euphoric, and presented a psychomotor activity which was almost equal to that seen in hypomanic states.

There were a number of well-defined cases of fear and compulsion neuroses. One patient, a girl aged 23, was in constant fear that she would commit suicide. She would always have to repress an impulse to jump out of a window, or throw herself before a train. Another patient, a man of 39, was in constant fear of being arrested, even though he felt positive he had done no wrong. A third man was afraid to stay home alone; he did not know what he feared, but felt safe if any one, even a child, would stay in the house with him. He realized how absurd his fear was, but maintained that he could not help or understand it. Another patient, a woman aged 33, could not fall asleep because she could not keep her thoughts away from people she knew who had died. Still another patient could not sleep because many details of the work he had done that day would repeatedly be reviewed.

Disturbance in memory, especially for recent events, was a common complaint. One patient, a student in accountancy, found that since his illness, in addition to defects in memory for recent events, he was having increasing difficulty in handling figures.

Changes in disposition were marked in three children. One, a boy of 10, had a mild attack of encephalitis in March, 1920. Previous to his illness his mother stated that she had great difficulty in managing this child because he was wilful, quarrelsome, and always up to some mischief; since his illness, he has become docile, obedient, and amiable, and never quarrels with his brothers and sisters as he had always done before his illness. Physical examination showed twitching in the

muscles supplied by the upper and lower branches of the right facial nerve and some residue in the other cranial nerves. He had gained twenty pounds; his mental reactions were slow and he resembled a mild type of Fröhlich's syndrome. Another boy of 13 was said, previous to his illness in April, 1920, to be an obedient, well mannered and well behaved child; he never had any difficulty in getting along with his companions. His attack of encephalitis was mild; however, his mother states that since his illness he has become extremely irritable and quarrelsome; he is wilful and difficult to manage. He has great difficulty in his studies and has fallen behind in his school work. He has acquired a spitting tic; every few seconds he has an irresistible desire to spit, and does so accompanying the act with an explosive grunt. He states that he cannot control this desire for more than a few minutes at a time. The third child began to steal little things, and to lie about everything he did; previous to his illness he had been a well behaved child. He has become mentally slow and has difficulty with his school work.

Insomnia was a complaint in forty-nine of the eighty-nine cases; this percentage is so striking that this symptom must be considered an undoubted sequel of encephalitis. Almost all of these patients stated that it required from one to two hours before they could fall asleep, and that until the early morning hours their sleep was easily disturbed; after the early morning hours and during the daytime their sleep was more profound. Some of the reasons given for this difficulty were pains in those patients who had suffered from the radicular type of encephalitis; others, the majority, thought it was due to the fact that they could not relax completely, and that their minds were constantly active. A similar insomnia was noted in many of these patients during the acute stage of their illness. This symptom, therefore, should not be considered a purely psychoneurotic manifestation. Many of the patients complained of excessive drowsiness throughout the day; some of them even fell asleep during their working hours, and were unable to hold their positions on this account. When they went to bed at night, however, sleep was difficult and in some cases even impossible.

Based on the publicity given the disease among the laity, many patients felt that they had acquired a certain importance in having passed through the dangers from it; these patients felt heroic, yet they strongly craved for sympathy and reassurance.

A more extensive analysis of the many variations in the disturbed psychic functions encountered among these patients has not been attempted. I think, however, that sufficient detail has been given to indicate that psychoneuroses and other forms of deranged psychic functions may follow this acute inflammation of the brain. Frank psychoses were not included in this study.

TREMOR AND IRREGULAR INVOLUNTARY MOVEMENTS

Fifty-two patients showed either tremor or some form of irregular involuntary movements. They were present in ten out of twenty-one cases eighteen months after the acute stage of the illness had passed; in eleven of twenty-one cases twelve months after; in twenty-nine of forty-two cases nine months after, and in two of five cases six months after the acute illness. Among these fifty-two patients, there were twenty-five who showed a fine or coarse tremor of either the lips, tongue, facial muscles, head, upper or lower extremities. These tremors closely resembled those seen in toxic states. The finer were like those of hyperthyroidism, and the coarser simulated those seen in chronic alcoholism. Most of the tremors were, as a rule, not evident when the parts were at rest, but became apparent when the parts were put into action. Ten of the fifty-two patients showed a tremor that was spontaneous, present while the parts were at rest, and as a rule limited to the distal parts of the extremity affected. Emotional stress caused exaggeration of the tremor, and it was said to be absent during sleep. This tremor was like the tremor of paralysis agitans. In three cases it was present in both hands; in three others it was present only in one hand; three patients presented tremor in the arm and leg of one side, and in one it was present in both arms and legs.

Tremors of the intentional type were noted in five patients. In three of these patients the tremor was present in both arms; in one the head and both arms were the seat of the tremor, and in the other all of the extremities were involved. These tremors were evident only when the parts affected were in action. They were characterized by large, coarse, irregular oscillations, with a variable range of movement which tended to increase as the goal was reached. They closely resembled the tremors seen in multiple sclerosis and cerebellar disease. One of the patients, in addition to the tremor, had nystagmus, scanning speech, and walked with an ataxic gait. She was, however, improving progressively.

Five patients had a rapid clonic twitch of the muscles supplied by one or more branches of the facial nerve. This twitching closely resembled the muscular response obtained when the nerve is stimulated with an electric current. It occurred every few seconds, was rapid, and always definitely anatomically limited to the muscles supplied by the particular branch involved.

Three of the patients had choreiform movements of the tongue, face or extremities. Three others had fibrillary tremor of the tongue; two of these were associated with a unilateral atrophy of the tongue. One patient had myoclonic movements of the perineal muscles one year after the acute illness.

DISTURBANCE IN ATTITUDE AND GAIT

Fourteen of the eighty-nine patients had disturbance in their attitude or gait, four patients—children—had the typical attitude and gait of paralysis agitans. The youngest was 7½ and the oldest 16 years. Five other patients showed similar attitudes and in addition walked with a hemiplegic gait. Loss of associated movements was present in five of these nine patients. Propulsion was present in four, and in one of these retropulsion was also obtained. Three patients walked with a spastic paraplegic gait and two walked with a right hemiplegic gait.

DISTURBANCE IN TONUS AND REFLEXES

Muscle tone was disturbed in seventeen cases. In four of these there was slight increase; in five there was moderate increase, but not sufficient to produce the “cog-wheel” phenomenon; in five others there was a marked increase, and the “cog-wheel” phenomenon was elicited. In two patients tonus was diminished in the muscles of the lower extremities, and one patient showed well-marked dystonia in the muscles of both thighs.

The deep reflexes were altered in twenty-seven cases; in eleven of these patients, the deep reflexes were greatly increased, but equally so on both sides. In fifteen the deep reflexes were unequal, being more active on one side than on the other. In one case, the right ankle reflex was absent, and the left was greatly diminished, while both knee reflexes were exaggerated.

The Babinski phenomenon was definitely present in six cases, and doubtful in one. In two of the six patients it was present on both sides, while in the four others it was present only on one side. Three of these patients presented clinically paralysis agitans features. In one of the patients there was associated with a bilateral Babinski sign, hypotonus at the ankles and loss of the right ankle jerk.

CRANIAL NERVE AND PUPILLARY DISTURBANCES

Some disturbance in the cranial nerves was present in fifty-seven of the eighty-nine patients. In thirty-six of these slight facial inequality was noted on one side; two others showed bilateral facial weakness. The slight disturbance in facial enervation in the majority of the cases seemed to be supranuclear in origin. Nine of the patients had both facial and external rectus involvement; two implication of the external rectus alone. Two had atrophy of the tongue and one had fibrillary tremor without atrophy. One patient had deviation of the tongue to the right, and one had paralysis of the soft palate on the right side. Five had some disturbance in the optic nerves during the acute stage of their illness. In this reexamination in only one patient definite changes in

the optic nerves were found; this girl had a temporal pallor on the right side and almost complete atrophy of the left nerve head. No optic neuritis was noted during the acute stage of this patient's illness. She also complained of diminution of hearing on the left side, but there were no objective signs of disturbed function present.

Incomplete ptosis of one eyelid was present in sixteen cases; on both sides in seven others. The pupils were unequal in twenty-six cases. Irregularity of the outline of the pupils was noted in five. Disturbance in the reflex to light or convergence was present in twenty-six cases; in ten of these there was a sluggish and incomplete reaction to light in both pupils; in seven others only one pupil was affected. In four patients there was a sluggish reaction to light on one side and complete loss of reaction on the other, with sluggish reactions to convergence in one or both pupils. Argyll Robertson pupils were present in five cases; in three of these it was present on one side only, and in two on both sides. One patient who had atrophy of the optic nerves, had what appeared to be on rough testing, a left homonymous hemianopsia, and the pupils were sluggish.

EPILEPSY

Three patients suffered from grand mal or petit mal attacks since their acute illness. The patient with the grand mal attacks had, since his acute illness in January, 1920, four seizures in which there was complete loss of consciousness for a period of from five to ten minutes. He also had a great many seizures in which he did not lose consciousness. The other two patients, both men, one 27 and the other 48 years of age, have been suffering since their acute illness from attacks of spasmodic contraction of the right side of the face. These attacks come on at frequent intervals, last from thirty to sixty seconds, and in the older of the patients are accompanied by vertigo, confusion and difficulty in speech which lasts from one to two minutes. If the men are standing when the seizure occurs they must grasp some near-by object to keep from falling. Any number of attacks occur each day.

OTHER RESIDUAL SIGNS AND SYMPTOMS

Headache and generalized pains in the various parts of the body was a most common complaint. The headaches presented no special characteristics; they were located over any part of the cranium, and were as a rule diffuse. Burning on the top of the head and behind the eyes was frequently an annoying sequel. Pains in the body and extremities was a frequent complaint of those patients who had suffered from the radicular form of encephalitis. Some of these patients stated that their pains were as severe as during the acute stage of their illness:

most of them, however, felt that their pains were gradually getting less severe.

Exophthalmos was noted in four cases. It was present on both sides, and was unaccompanied by any other signs of hyperthyroidism, with the exception of tremor of the hands. Tremor of the hands, however, was present in so many of the patients that it could not definitely be attributed to hyperthyroid activity in these patients.

Increase in weight sufficient to be noted by the patient or his people was found in fifteen cases. In thirteen of these the amount gained above the best previous weight was 15, 15, 17, 20, 20, 25, 25, 35, 38, 45, 48, 50 and 95 pounds. In two the exact amount gained was not definitely known. Many of these patients showed some other manifestations of disturbed pituitary function.

PROGRESSIVE CASES

Seven of the patients gave definite evidence of progression in their disease at the time they were examined. Five of these were of the paralysis agitans type. They showed the typical attitude, gait, tremor, rigidity, and restlessness seen in this disease. In some of these, disturbance in associated movements was also present. One of the other two patients, a man 30 years old, has gained ninety-five pounds in weight since his illness sixteen months ago; he is mentally sluggish and always drowsy; six months ago fibrillary tremor and atrophy of the left half of his tongue were noted. He is progressively getting worse. The other patient was taken ill in November, 1919. At the time her physical examination revealed bilateral facial weakness, tremor of the tongue, intention tremor and rigidity with cog-wheel phenomenon in both arms; her attitude and gait were that of a patient with paralysis agitans. In December, 1920, reexamination showed that she walked with a peculiar gait, not unlike that seen in progressive torsion spasm; there were choreiform and choreo-athetoid movements of the face, neck, shoulders and lower extremities. There was a constant uncontrollable grinding of her teeth. While seated, her pelvis and lower extremities were constantly being twisted by involuntary spasms of the muscles. Her pupils were irregular and almost completely immobile in their reactions to light and convergence. There was slight flattening of the facial folds on the right side. Dystonia was present in the muscles of the lower extremities. The deep and superficial reflexes were present and equally active on both sides. The Babinski sign was not elicited.

The majority of the other patients admitted that they were gradually improving. Even though they still had many complaints, they were much better and the symptoms were far less severe than they had been at the time the patients were in the hospital.

MORTALITY

Among the 145 patients admitted to the hospital twenty-nine died, giving a mortality rate of 20 per cent. Notice was received of the death of one other patient after she left the hospital.

SUMMARY AND CONCLUSIONS

Sufficient time has not yet elapsed since the acute illness, nor is the number of cases in this study large enough, to warrant drawing absolute conclusions as to what the ultimate prognosis will be in these patients. There are, however, a number of striking facts that might be emphasized.

1. Psychic functions in some form or another were disturbed in 55 per cent. of these patients.

2. Insomnia was present in 55 per cent. of the cases.

3. Tremor and irregular involuntary movements were found in 58 per cent. of the cases.

4. The deep reflexes were altered in 30 per cent. of the cases, and tonus in the muscles was disturbed in 18 per cent.

5. The cranial nerves showed residual signs in 64 per cent. of the cases.

6. Pupillary disturbances were found in 30 per cent of the cases. Five patients had Argyll Robertson pupils.

7. About 8 per cent. of the patients gave signs of progression at the time they were examined.

8. The mortality among the 145 patients admitted to the Mount Sinai Hospital was 20 per cent.

From these findings one might venture this tentative prognosis: Probably less than 20 per cent. of the patients who become ill with epidemic encephalitis die during the acute stage of the illness, as usually only the most severe cases reach the hospital. Of those who survive the acute stage, about 10 per cent. may develop a progressive disease of the central nervous system. The remainder will make a good functional recovery in from six to twenty-four months, with the probability of progressive approach to the normal after that period.

News and Comment

CENTENARY OF BLOOMINGDALE HOSPITAL

The hundredth anniversary of the opening of the Bloomingdale Hospital will be celebrated on May 26. The exercises will include addresses by Dr. Pierre Janet of Paris, Dr. Richard G. Rows of London, Dr. Lewellys F. Barker and Dr. Adolph Meyer, Baltimore. The hospital is the department of nervous and mental diseases of the New York Hospital which, since it was opened in 1792, has made provision for the treatment of persons suffering from mental disorders. As early as 1808 a separate building was provided for these patients at the general hospital at Duane Street and Broadway, New York. In 1821, the department was removed from the city to a point on the Bloomingdale Road which is now the location of the library of Columbia University, and was given its present distinctive name. Since 1894, it has been located at White Plains.

Abstracts from Current Literature

LA MICROGLIA Y SU TRANSFORMACION EN CELULAS EN BASTONCITO Y CUERPOS GRANULO-ADIPOSOS (MICROGLIA AND ITS TRANSFORMATION INTO ROD CELLS AND GRANULO-ADIPOSE BODIES). P. DEL RIO-HORTEGA, Arch. de Neurobiol. 1:171 (June) 1920.

The author summarizes the article thus:

1. Relation between microglia and the third element (of Cajal).
2. Characters of normal microglia: (a) technic; (b) morphology of the microglia; (c) morphologic variations in normal states; (d) distribution and relations of the microglia.
3. Abnormal characters of microglia: (a) first morphologic changes; (b) the problem of rod cells; (c) formation of rod cells; (d) character of the rod cells; (e) problem of granulofat cells; (f) formation of granulofat cells.
4. The probable nature of the microglia.

The author gives a résumé of the views of Alzheimer, Held, Fieandt, Jacob, Lugaro, and others, who have denied the existence of a so-called third element of the neuroglia, and of Bevan, Lewis, Nissl, Robertson, Bonome, Schaper, Rosenthal, and others, who have considered the third element as undifferentiated glia without dendrites, and have thought that they had observed transitional forms between the adendritic bodies (third element) and dendritic glia cells. He then gives Cajal's conclusions as to a veritable third element, at length, resulting in the classification of the third element into two groups, subdivided into several subgroups; the two groups being (a) cells of the gray matter, perineural satellites not neuroglial, and disseminated apolar cells; (b) apolar cells of the white substance, either isolated or arranged in rows.

The author then passes to his own methods of research which have been carried out on the brains of monkeys, rats, dogs, cats, rabbits, oxen, goats and sheep; on linnets, pigeons and hens; lizards, newts, snakes, chameleons and turtles; toads and frogs; and many fish and above all on human brains of all ages and of people dying of ordinary diseases.

In general, microglia is described as consisting of very small corpuscles provided with large, ramified expansions. It is diffused throughout the nervous tissue, but differs in character in varying localities. It differs from neuroglia in not possessing the characters of protoplasmatic glia, reticulum, gliosomes, nor those of fibrous glia, differentiated fibrils. Centrosomes and Golgi's net are also absent in microglia.

Morphologically the microglial cells are:

- (a) Monopolar with dendrites ramified not far from the body.
- (b) Bipolar with two thick dendrites with many right-angled collaterals, the principal dendrites often ending far from the cell in a plume of very delicate branches, often double nucleated.
- (c) Multipolar, most numerous of all and offering great variety of form.

The microglia is present in all parts of the brain in greater or less quantity, but much more in the gray matter than in the white.

In the gray matter the greater number of neuronal satellites are probably microglial. The vascular satellites are also greater in number in the gray than

in the white substance. These vascular satellites do not enter into intimate relations with the vessels as in the glial feet of neuroglia. Microglial cells are also seen as satellites of neuroglial cells.

These morphologic characters are identical in man and the vertebrates studied, differing in this manner from neuroglia which shows marked differences in the different groups of vertebrates.

The first pathologic change which takes place in microglia is an increase in volume, especially of the dendrites; sometimes hypertrophy of all the microglia occurs. This change is seen in man, especially in acute and subacute meningitis, meningo-encephalitis and uremia, but seen to a certain extent in many other pathologic states. Shortening of the dendrites occurs coordinately with their thickening; argentophile granules are seen, but none colored by scarlet.

The author considers the various theories of the origin of rod cells, describes them as characterized by a long, rectilinear, curved, round, ovoid or fusiform nucleus, pale protoplasms, orientation almost always parallel to blood vessels and perpendicular to the surface of the cortex. He notes atypical forms. He considers the rod cells as phagocytes and goes into detail as to their formation from emigrating microglial cells, with the same minutia as he studies the history of the granular cell and finally describes the formation of granular cells from the microglia.

His conclusions are:

1. Besides the neuroglia a third type of cells exist whose character and origin have given rise to much argument.
2. Two forms of neuroglia have been isolated, one protoplasmatic with short dendrites especially proper to the gray matter, the other fibrous with long radiations proper to the white substance.
3. The third element has been considered as an interstitial element characterized by the absence of prolongations which would take the elective glial stains.
4. In such cells, apparently apolar, two varieties have been recognized, one with a large clear nucleus, the other with the nucleus small and dark colored.
5. The author's observations confirm the actuality of both types of cells mentioned in the foregoing, but of a character entirely different to what has been assumed hitherto. The two types are (a) interfascicular glia and (b) microglia.
6. The interfascicular glia, which is situated by preference between the nerve fiber bundles of the white substance, although also accompanying nerve cells and blood vessels, is characterized by a large vesicular nucleus, a round or polyhedral body, by its epithelial appearance and by scanty, long, filiform prolongations, little branched.
7. The microglia is seen throughout the nervous tissue, more abundant in the gray than in the white matter, and is characterized by its small dark nucleus surrounded by little protoplasm, and by its long, tortuous expansions which are much ramified and provided with lateral spines. Neither gliosomes nor gliafibrils are seen in its obscurely reticulated protoplasm, but pigment and lipid granules are frequently seen in its substance.
8. According to its situation, the microglia is classified as a neuronal, vascular and neuroglial satellite.
9. No morphologic or structural differences exist in the microglia of different vertebrates.

10. This constancy of character is due principally to the fact that the microglia is subjected to the general disposition of the nervous system between the interstices of which the prolongations of the microglia are inserted. Therefore, the forms seen are due to adaptation to the surrounding mediums.

11. The instability of form of the microglia is seen more in embryonal development and in the pathologic conditions than in adult and normal brains.

In their evolution the microglial corpuscles pass from a rounded to a stellate form, and in regression they tend to recover the original form.

12. Judging from its action in pathologic conditions, the microglia is especially fitted for phagocytic action on the degenerative products of disintegrating nerve cells.

13. When the microglia is put into movement and enters on its phagocytic action in consequence of some disturbance of the nervous system, its form is modified little by little, and the microglia is transformed into rod cells and granulo-adipose cells.

14. The long bipolar cells (rod cells) appear only when the microglia extends parallel with fiber bundles, or nerve cell dendrites. Ordinary methods of staining show these cells only in pathologic processes of slow development. In acute processes the bipolar types regress to the multipolar type or are transformed into the more or less rounded granular type.

15. In order to become rounded (granular cells) the microglia cells must be freed from the pressure of the surrounding structures, as by liquefaction or rarefaction, as well as filled with degenerative products.

16. The forms which the microglia takes on in pathologic processes are similar in form, structure and coloring qualities to those of the emigrating connective tissue corpuscles in abscesses, tumors, etc.

17. There are many reasons for considering the microglia mesodermic in origin and, if confirmed, the name mesoglia would be justified.

18. The microglia is, therefore, a third element of the nervous centers. The interfascicular glia appears to be a third type of neuroglia of epithelial origin.

The author notes that during the preparation of the present article he has made important observations to be published fully, later, which tend to prove the mesodermic origin of the microglia.

As usual in the work of the Spanish school, the illustrations are very profuse, excellent and conclusive.

GURD, Ann Arbor, Mich.

DIE PLEUS CHOROIDEI BEI ORGANISCHEN HIRNKRANKHEITEN
UND BEI DER SCHIZOPHRENIE (THE CHOROID PLEXUS IN
ORGANIC DISEASES OF THE BRAIN AND SCHIZOPHRENIA).
SADAMICHI KITABAYASHI, Schweiz. Arch. f. Neurol. u. Psychiat. 7:1, 1920.

Comparatively little work has been done on the study of the choroid plexus in the organic diseases of the brain, and its physiologic significance is comparatively unknown. The present contribution is a continuation of the study of the choroid plexus, several reports of which have been abstracted in these columns. Previous investigations of the brain of schizophrenic patients have shown that the choroid plexus is subject to characteristic alterations in this disease. Degeneration productions of various kinds, which cannot be more closely identified, are numerous. Some of these penetrate the ependymal wall into the subependymal structures and even into deeper tissues of the brain, probably as a result of abnormally low resistance of the ependyma.

The present report was based on the study of three groups of cases. The first group is made up of five patients whose ages ranged from 11 months to 55 years, whose brains were assumed to be practically normal. The second group of eight patients represents those who had chronic diffuse or localized lesions of the central nervous system without schizophrenic manifestations. The third group is composed of patients who had schizophrenia.

Kitabayashi studied not only the choroid plexuses themselves, but also the adjacent ependymal walls. The material was fixed in formaldehyd, dehydrated with alcohol, and embedded in celloidin; sections 15 microns in thickness were stained with hematoxylin, van Giesen, toluidin blue, and carmin. Some of the material was embedded in paraffin.

The normal plexus was found to differ somewhat in the microscopic appearance, depending on the age. In children and young persons, the vessels of the plexus are very delicate throughout; the connective tissue is small in amount, very delicate in structure, and free from large spaces. The glandular cells have a grapelike arrangement near the base of the villi and like a string of pearls along the free portion, there being but one row of cells here. The protoplasm is finely granular throughout, free from definite tigroid elements, fills the cells completely, and contains a well defined, deeply staining nucleus which contains a nucleolus. Vacuoles are not seen. There are no colloidal masses, no hyaline, and no calcium deposits anywhere. The appearance of the plexuses in the different ventricles is essentially the same.

The above description, in the main, applies to all normal cases; in old persons, however, there are slight modifications. In a patient 30 years of age, the perivascular spaces were still small and delicate in structure as compared with those found in older persons. Only occasionally was a nucleus seen that was somewhat shrunken. Here and there an area was found in which the connective tissue was somewhat thickened and showed slight hyaline degeneration. At the age of 35, in addition a few calcium granules were noted. These were numerous in the perivascular spaces of a man 55 years of age. The plasma and nuclei of the glandular cells were slightly atrophic.

Since the patients of the second group showed changes that were not uniform, it is necessary to review them individually. The first was a premature baby of about 7½ months, who had hydrocephalus and died of convulsions. Necropsy revealed, in addition to the hydrocephalus, microgyria and almost complete absence of the corpus callosum. The villi of the choroid plexuses were small (microplexia). The cells themselves had the appearance of atrophy, possibly the result of pressure. In the connective tissue spaces, a few albuminoid granules were noted, which is unusual for normal children of the same age. The ependyma showed an alternating atrophy and hyperplasia; the subependymal tissue was hyperplastic, probably the result of pressure or penetration of noxious substances contained in the spinal fluid.

The second patient was a taboparetic, 42 years of age, without hallucinations and delirium, who died suddenly in a status epilepticus. The plexus showed little that was characteristic; there was slight swelling of the cells and a few small vacuoles were noted. There was a slight exudate which, however, was entirely free from the granules that are so characteristic of schizophrenia.

The third patient of this series was a man of 61 years, who had had right-sided jacksonian epilepsy for a number of years, and headaches. Examination revealed choked disks, a right-sided hemiplegia, dysarthria and motor aphasia. There were no marked psychic alterations. A decompression operation fol-

lowed. Death occurred suddenly at the end of seven years. Necropsy revealed an angiosarcoma in the left central convolution. The plexuses showed a dense increase in the connective tissue of the perivascular spaces and some hyaline degeneration of the plexus cells.

The fourth case was that of a teacher, 72 years of age, who, following fracture of the tibia, developed a marked psychosis with dementia, confabulation, delirium, hallucinations, complete disorientation—on the whole, a manic picture of several months' duration. The plexuses revealed changes ordinarily noted in senile patients, namely, an increase in connective tissue and deposits of calcarious concretions; in addition, there were findings not characteristic of senility, such as marked degenerative changes of the plexus cells, which were swollen and showed ameboid processes, vacuolization and desquamation. The exudate in the intervillous spaces contained albuminoid granules that could be traced into the subependymal tissue where they doubtless acted as foreign substances. The writer believed that there was probably a close relationship between the pathologic findings and the clinical manifestations.

The fifth case was that of a weaver, 65 years of age, in whose case a diagnosis of an organic psychosis with manic-depressive features was made. The patient showed increased psychomotor activity with impulsive acts, visual and auditory hallucinations, and sitophobia. The clinical picture approached that of schizophrenia. The villi of the plexuses were markedly degenerated; there was sclerosis of the cells, reduction in size of the nuclei, desquamation of entire rows of cells, massive concretions within the villi, and numerous albuminoid masses that could be followed into the subependymal tissue. There was also a heterotopia of the vermis. The writer thought that here also the anatomic findings might explain the psychic processes.

The sixth patient was a porter, 47 years of age, who was a chronic alcoholic, and who had suffered a fracture of the spine. He had hallucinations, delusions of persecution and marked disorientation. He died of pneumonia. The vessels of the villi were degenerated; there were perivascular extravasation of the blood and atrophy, sclerosis, swelling, desquamation and vascularization of the choroidal cells. The writer believed that the parenchymatous and hyaline changes of the capillaries could be attributed to alcoholism; capillary thrombosis and blood extravasation followed the vascular degeneration. The exudation between the villi might have resulted from fever.

The seventh patient was a 70 year old man, who suffered from arteriosclerotic dementia with epilepsy, illusions and delusions of persecution. At necropsy, senile changes were noted in other parts of the body and evidenced in the choroid plexuses by sclerosis, tortuosity and thrombosis of the arteries, and marked enlargement of the perivascular spaces, which contained dense calcarious concretions. These changes may be attributed to the arteriosclerosis. There was also desquamation of the cells, the latter being in part atrophic and sclerotic, and in part swollen and vacuolated. The connective tissue contained numerous albuminoid bodies, some of which penetrated into the subependymal structures.

The last patient was a man 53 years of age, a congenital deafmute and an idiot. The plexus cells were moderately atrophic—some were strikingly small, others sclerotic or swollen and still others desquamated. There was definite connective tissue increase about the blood vessels; hyaline degeneration and calcified granules were noted in the villi. These findings were probably the result of age. On the other hand, in the spaces between the villi albuminoid

bodies were noted which also penetrated into the subependymal tissue. The vermis was heterotaxic; the ventral acoustic ganglion and the tuberculum acusticum were greatly degenerated, which probably explained the deafness.

The article will be continued.

WOLTMAN, Rochester, Minn.

LE HOQUET EPIDEMIQUE, FORME SINGULTEUSE DE L'ENCEPHALITE EPIDEMIQUE (EPIDEMIC HICCOUGH, A SINGULTOUS FORM OF EPIDEMIC ENCEPHALITIS). J. LHERMITTE, *Presse méd.* **28**:916 (Dec. 18) 1920.

The epidemics of hiccough noted by Boerhave at the hospital of Haarlem, that of the convent of Monterrey (Spain), also those reported by the medieval historians and attributed to demon-possession, formerly found a fully satisfactory explanation in the theory of hysteria. Recent epidemics of hiccough have proved that even if certain cases do arise from suggestion or psychic contagion, there are others for which another explanation must necessarily be found.

Von Economo reported the first appearance of epidemic hiccough at Vienna in the winter of 1919-1920, as follows: "Several weeks before the appearance of this epidemic of encephalitis in Italy (January, 1920), there was a small epidemic of singultus in and near Vienna. Numerous individuals were suddenly seized with painful attacks of hiccough, without any preliminary symptoms of illness; these attacks lasted hours and even whole days, were not affected by any treatment, and ceased spontaneously at the end of a few days. I saw a similar case that lasted an entire month, the patient having only a few hours of rest each day. This disease had no serious sequelae."

One month after this epidemic of hiccough was over the first cases of myoclonic encephalitis appeared, imitating the variety that had just undergone a violent exacerbation in Italy. The illness commenced with general malaise, accompanied by vague rheumatic pains and often by severe neuralgias. Then an occupation delirium appeared, with visual hallucinations, and finally clonic convulsions of the abdominal muscles, and hiccough. At this point a state of choreiform agitation supervened, followed by myoclonic contractions or fibrillary twitchings, not interrupted by sleep.

In January and February of 1920, Dufour and R. Bénard described a number of cases of hiccough in and near Paris. Typical cases cleared up about the fourth day; one terminated in myoclonic convulsions and death. Staehelin's studies at Basle revealed the successive development, first, of an epidemic of transitory ocular paralyses, then of an epidemic of hiccough, finally of an outbreak of numerous cases of epidemic encephalitis. In November, 1920, epidemic hiccough reappeared at Paris.

In a simple hiccough the abdominal wall is passively lifted, but its muscles do not exhibit spasmodic contraction. In epidemic hiccough the clonic spasms may not be so closely limited to the constrictors of the glottis and of the diaphragm, but may extend to the abdominal muscles and even reach segments of musculature whose rôle is entirely independent of the respiratory function. Clonic contractions added to the spasmodic shocks of hiccough occur most often in the muscles of the back, the nucha, or the limbs. Different attitudes result, as backward bending of the trunk, bowing of the head, flexion of the extremities, which vary with each patient, and in the same person are subject to modifications according to the changing conditions in the nervous system. In rarer cases, the hiccough is associated with spasmodic contractions limited to the abdominal muscles of one side. Achard recently

demonstrated radioscopically a strictly unilateral contraction of the diaphragm in an epidemic hiccough case.

Pain is uncommon. Phonation and deglutition are markedly disturbed. Patients adopt various psychic attitudes: some are greatly distressed and give up all social activity; others "rebel, and while excusing themselves to their associates for this inopportune ailment which they consider ridiculous, make no change in their daily habits." The rhythm of hiccough ranges from six to fifteen contractions per minute, which go on without intermission for hours, sometimes for whole days. The total duration is three or four days, ordinarily, but may be much longer. The onset may occur without any prodromal symptoms, but a diligent examination or searching anamnesis will often discover general malaise, chilliness and headache, accompanied by slight elevation of temperature to 99 or 100 F. Mild emotional disturbances are often detectable. Visual functions, deep and superficial reflexes, and general muscular tone show no abnormalities.

Epidemic hiccough constitutes a syndrome whose elements, outside of the phrenoglottic myoclonus, are necessarily elusive, fleeting, and variable. It is differentiated from hysterical and even more from simulated hiccough by the fact that certain of its characteristics cannot be reproduced by a normal subject. The frequency of hiccough in abdominal and peritoneal disease has led to a number of surgical operations on patients with epidemic hiccough. Typical lesions of acute encephalomyelitis have been found at necropsy.

Quoting a number of other European and American observers on the frequency of hiccoughs in epidemics of unquestioned "encephalitis lethargica," Lhermitte believes the epidemiologic and clinical evidence is conclusive that epidemic hiccough is only a masked form of epidemic encephalitis. The pathogenesis remains unknown and the therapeutics unreliable. Treatments for hiccough fall into two groups—drugs that counteract bulbospinal and vagophrenic hyperexcitability (belladonna, atropin, cocain, morphin, bromid, camphor, oxygen), and physical means of exercising an inhibitory action on nerve centers in a state of reflex excitement (a dozen of these are mentioned). Their diversity denotes their inadequacy.

HUDDLESON, New York.

BEITRAGE ZUR KLINIK UND PATHOGENESE DER LUMBAGO
(CONTRIBUTIONS TO THE CLINICAL STUDY AND PATHO-
GENESIS OF LUMBAGO). RUDOLF BRUN, Schweiz. Arch. f. Neurol. u.
Psychiat. 7:63, 1920.

Although lumbago is a subject of great practical interest, the serious study of it has been neglected until a comparatively recent date. Even at the present time this banal disturbance is little understood. The clinical findings have been almost uniformly negative.

In all probability, "lumbago" has numerous and diverse pathologic bases. Its tentative division under the three heads of myogenic, osteo-arthrogenic and neurogenic, probably represents impressions and theories rather than well-founded clinical diagnoses.

The myogenic form has been further subdivided. The assumption is that the majority of these cases are rheumatic in nature; this would also apply to most cases in which the symptoms followed an injury, the idea being that these muscles had been rendered more susceptible to traumatism by a low grade myositis, the symptom of pain becoming manifest only on the occasion of an injury. The history of a previous rheumatic condition in some other location

strengthens the diagnosis. Many writers share the opinion that most cases of lumbago represent recurrences of a subacute or chronic lumbar myositis. According to Gelpke, secondary infection of the traumatized muscles explains the usual chronic course of the disease.

The second division includes the actual muscle trauma, such as rupture, tearing of the ligaments and fascial hernias.

The osteo-arthrogenic type is probably rare.

The neurogenic type is supposed to originate from compression of the sensory nerves. The frequent association with sciatica has been explained as a spreading secondary perineuritis.

Brun reports twelve cases of so-called "lumbalgia," in which in each instance objective findings could be demonstrated. Of fifteen cases seen in the Zurich neurologic clinic between 1915 and 1919, only one in five gave negative findings. According to Pometta, one of the best students of the subject, traumatic lumbago should heal within from five to eight days. In these patients, however, the progress was eminently chronic, extending over months and even years. He could almost uniformly demonstrate some abnormality in the region of the lumbar musculature, particularly the erector trunci and the sacrospinalis muscles. In eleven of the twelve cases, inspection and palpation alone gave positive findings. In two cases a transverse groove was found, which was interpreted as a rupture of the muscle; this could be demonstrated more clearly by voluntary movement or by faradic stimulation when it was noted that the upper portion of the ruptured muscle retracted strongly upward while the lower portion did not respond. In the ten remaining cases the lower insertion of the sacrospinalis muscles appeared definitely depressed, atrophic, inelastic, tender and in every instance gave abnormal electrical responses. Irritability to the faradic current was diminished or did not result in mass contraction. Of seven cases tested with the galvanic current, only two gave normal responses, while the remaining five showed a partial reaction of degeneration. Abnormality of the vertebral column was noted in some. Tuberculosis of the spine was disclosed by the roentgen ray in a patient whose case had for a long time been diagnosed as lumbago; the roentgen-ray findings in the others were negative. Tenderness of the spine was not uncommon. Scoliosis, when present, was usually directed toward the healthy side. In several cases, there was a spasmodic reflex fixation of the lumbar vertebrae on bending. Hyperesthesia and hypesthesia were noted in the area supplied by the posterior roots of the lumbar and sacral nerves, the ileo-inguinal and the sciatic. Reflex irritability of the sympathetic supply was evidenced by cutaneous flushing on slight irritation. In three cases there was an associated neuralgia of the ileo-inguinal nerve, in one of which an associated spermatorrhea improved coincident with the recovery from the lumbago. Tenderness on pressure over points at which these nerves pierce the fascia was common. The lower abdominal reflexes were often reduced while the cremasteric reflexes were increased. In all but one patient there was referred pain along the ipsilateral sciatic nerve, although the characteristic signs were usually absent.

Brun reviews somewhat extensively the sympathetic innervation of muscles and the difference in metabolism of the two components of muscle fibers as described by Boeke, Pekelharing, de Boer and others. Their work is applied to the question of lumbago in so far as he assumes that practically all of these patients are neurotic; their sympathetic innervation is therefore altered, and in such a manner that the musculature loses its ability to with-

stand sudden stresses, thus becoming more fragile and susceptible to rupture. Ischemia also plays a part in this.

He calls attention to the fact that a nerve may easily be injured in its inter-vertebral course by a transient subluxation of the spine, which may at once correct itself, while the injury to the nerve persists. Extension to the sciatic and ileo-inguinal nerves usually does not occur until several weeks or months after the initial trauma. This radiation, he states, takes place through the spinal ganglions and is dependent on the increased irritability of the sympathetic cells as well as on over-irritation of the remaining uninjured fibers.

The frequent chronicity of the disorder and its association with a general neurosis can be demonstrated by psychoanalytic methods, to rest on a sexual basis, the lumbago becoming the peace offering onto which compunctions of conscience arising from onanism and impotence can be heaped.

Brun's conclusion is that lumbago is usually due to perineuritis of traumatic origin and seldom rests on a toxicinfectious basis. He emphasizes the importance of a careful study of the individual case.

WOLTMAN, Rochester, Minn.

INTELLIGENCE AND PSYCHOSIS FROM THE PSYCHOLOGICAL
LABORATORY OF McLEAN HOSPITAL, WAVERLEY, MASS.
F. L. WELLS and C. M. KELLY, *Am. J. Insan.* **77**:16, 1920.

One hundred and two patients, both men and women, ranging in age from 16 to 75 years, suffering from mental disease, various psychoses being represented, were examined according to the Stanford Intelligence Scale, and the results were tabulated according to diagnostic groups. Analysis showed: 1. Reduction of intelligence to subnormal was not a necessary accompaniment to grave mental disorder. Seventy per cent. of the patients had intelligence quotients above 70 and except for the organic group, over one half presented intelligence quotients above the level to which independent adjustment is for reasons of intelligence no longer possible, and some were considerably above the normal level. 2. Little diagnostic importance could be attached to intelligence quotients, but the organic cases showed the greatest reduction, being the only cases in which intelligence defect showed definite association with the psychosis. 3. The greatest amount of "scattering" was found in the organic group, less in the dementia praecox and least of all in the manic-depressive group, but the difference between the groups was small. A few single tests were the only differential features. For example, absurdities (X-2), designs (X-3), reading and report (X-4) were never passed above age, but failed with similar frequency in the three groups. The superior plan, ball and field (XII-3), failed below age over twice as often in the dementia praecox group as in the manic-depressive or organic group, while repetition of five and six digits backwards (XII-6, XVI-5) resulted in nine failures below age, two successes above in the manic-depressive and organic groups with one failure below age and eight successes above in the dementia praecox group. It was also noted that several of the tests appeared to be differently weighted for the insane, that is, they did not have the same value for the psychotic as for normal persons of a given mental age. Designs (X-3), reading and report (X-4), and ball and field, superior plan (XII-3), were much more difficult for these subjects, showing a marked tendency to failure below mental age, while vocabulary and formal memory tests were the least difficult and the most frequently passed above mental age.

Some criticism of detail was offered, principally defects inherent in the scale, especially true when used for patients of this class—but the scale was not constructed for this group. Among the most noticeable were the childish phraseology of many of the tests, problems which presented situations incongruous with adult experience or of limited application because of local and geographic differences. Several of the tests lent themselves most readily to coaching and were therefore found just that much less valuable. Duplication of digits and sequences in the number series for memory tests were objected to on psychologic grounds. Other tests allowed scope for perseveration. Lastly, the emotional setting of certain of the tests was disturbing to some patients and influenced results, while others allowed for perverted responses, most conspicuous in the dementia praecox group. It was further noted that "intelligence" as measured by the scale was not all-inclusive in that ability to deal with ideas is emphasized while no cognizance is taken of ability to deal with things or make adjustments to other persons, both equally important in "real" action. Intelligence in this limited sense is neither the sole nor the chief factor in practical mental adjustment but is essential in some degree. In so far as the scale measures ideational capacity, it was found to be satisfactory and practical but with the psychotic groups the failures of adjustment for the most part are not in the sphere of ideas or things but in the more instinctive and affective adaptations to other members of society. Similar intelligence quotients, therefore, appeared in extremely differing personalities and normal ones in the presence of grave mental imbalance.

PERKINS, Detroit.

TRAITEMENT DE LA SYPHILIS NERVEUSE PAR LES INJECTIONS NOVARSENICALES A PETITES DOSES REPETEES ET PROLONGEES (TREATMENT OF SYPHILIS OF THE NERVOUS SYSTEM BY PROLONGED COURSES OF FREQUENT SMALL INJECTIONS OF NEO-ARSPHENAMIN). J. A. SICARD, *Presse méd.* 28:281 (May 8) 1920.

One of the common neurologic schemes of intensive arsenical medication is a course of from four to six weekly intravenous injections, often in ascending doses, as 0.3, 0.45, 0.6, 0.75 gm. Some workers have tried reducing the dosage and lengthening the interval. This paper presents arguments against these plans, and sets forth the advantages of the following one.

A neo-arsphenamin product ("novarsénobenzol," "sanar," and "galy") are mentioned) is injected, either intravenously or subcutaneously. Subcutaneous doses do not exceed 0.15 gm., dissolved in 1 c.c. of sterile distilled water. The preferred site of injection is the upper third of the thigh; little pain is experienced and no complications need be feared. Some patients have received without ill effects, as many as sixty daily subcutaneous injections, alternating between the right and left subtrochanteric areas.

The total dose of neo-arsphenamin for general paralysis reaches an average of from 28 to 30 gm. in a year, divided into 9 or 10 gm. for a four month period, 0.15 gm. being given in daily intravenous injections. For progressing tabes, from 20 to 25 gm. are given over a year's time, divided into 6 or 8 gm. for a four month period, two subcutaneous injections of 0.15 gm. and one intravenous of 0.3 gm. being given weekly. For spastic paraplegia, either the tabetic or the paretic outline is followed. The total quantity of drug in any course is reduced for appropriate indications.

This method dispenses altogether with mercury and iodid preparations, and avoids toxic effects peculiar to them. It is claimed that it also avoids the more serious accidents sometimes associated with other methods of neo-arsphenamin administration. Four milder types of reaction are observed: erythema, loss of the Achilles' reflex, late jaundice and slight transitory nitrogen retention.

Two varieties of arsenical erythema are distinguished. One appears early, in 6 or 8 per cent. of cases, after 0.5 to 3 gm. of neo-arsphenamin have been administered; it disappears readily and is generally insignificant. The other appears late and constitutes positive evidence of arsenic saturation. It will progress rapidly through severer stages of skin and constitutional disturbance, unless the drug is discontinued immediately. Treatment is not to be resumed for eight or ten weeks thereafter. This erythema shows an incidence of 2 or 3 per cent.

Disappearance of the Achilles' reflex is discussed at length. This phenomenon attains the remarkable incidence of 60 per cent., but it must be remembered that a course of treatment totals from 7 to 10 gm. of neo-arsphenamin. Incipient objective signs can be detected after 3 or 4 gm. have been administered, and their evolution follows until both ankle jerks disappear completely, during about the sixth or seventh week of the course. As a rule, no subjective weaknesses nor sensory disturbances are experienced by the patients, but paresthesias of the soles and great toes appear occasionally. Several cases have been observed over one and one-half years, and no abolished ankle jerk has reappeared. However, no loss of muscular power supervenes. Syphilitic spastic paraplegias are especially benefited by this fact. A diminishing ankle jerk may go on to complete disappearance one or two weeks after medication ceases. One patient of 200 treated developed a polyneuritis after receiving 4 gm. of neo-arsphenamin.

Jaundice appears in 2 per cent. of cases. It is considered to be of toxic arsenical origin, and calls for discontinuing medication. Uneventful recovery takes place three or four weeks later. Nitrogen retention is of similar import.

Blood and spinal fluid Wassermann reactions, as well as clinical symptoms, are said to be most effectively influenced by this method. No objective signs of intoxication have been discovered in visual, auditory or other sensory fields. The theoretical possibility of producing an arsenic-resisting strain of spirochetes is set aside as already disproved by certain Pasteur Institute work (not cited in detail). Final conclusions are stated moderately: "Comparing equal quantities of neo-arsphenamin injected throughout equal periods of time, one finds that small doses given daily or every other day afford a degree of certainty and safety that cannot be claimed for weekly injections."

HUDDLESON, New York.

ACUTE PSYCHOSES ARISING DURING THE COURSE OF HEART DISEASE. DAVID RIESMAN, *Am. J. Med. Sc.* **161**:157 (Feb.) 1921.

This is an interesting presentation, from the standpoint of the internist, of the acute psychoses and psychotic symptoms, arising in heart disease. Probably the most common mental syndrome is a state of irritability with a substratum of emotional depression which often occurs in association with cardiac decompensation. A frequent cardiac-psychotic symptom is an hallucination (?) of sight or hearing, which appears in the course of aortic disease. "The hallucinations are to be looked upon as misinterpreted sensations caused either

by anemia or by venous stasis of definite brain centers, those connected with sight or hearing, or by circulatory or trophic disturbances in the corresponding peripheral end organs." If the author's theory is true and we are really dealing with a misinterpreted sensation, it would be better to speak of an illusion and not an hallucination.

Riesman also calls attention to the period of confusion noted just prior to, or just after, sleep in patients with myocarditis and auricular fibrillation; to the mental excitation and marked disorientation in elderly patients with fibroid myocarditis, which is of grave prognostic import; to the abrupt maniacal out-breaks, which occurred in acute pericarditis and in advanced decompensation of myocardial origin, and to the paranoid delusional trend in conjunction with aortic disease.

There are a number of etiologic possibilities: In a person whose heredity is heavily charged, the psychotic manifestations may be entirely independent of the cardiac pathology; kidney disturbance, and more particularly, uremia may be accountable; acidosis may be the precipitating agent or unwise digitalis therapy may "by disturbing cardiac rhythm through its action on the conducting mechanism, still further impair an already inadequate circulation" and thus produce mental symptoms. In the absence of any of these factors "we may assume some direct disturbance of the cerebral circulation, affecting the higher centers." In this connection Dana's¹ theory of "hypo-function of synapses" may condition the abnormal psychotic response to the somatic disease.

From the standpoint of the psychiatrist, our knowledge must be said to be still in the formative stage. We must first be given more light on the chemistry of heart disease and indeed of somatic disease in general. Clinically, it is probably true that the more widely the mental symptoms depart from the classic psychoses, the more readily and accurately may they be attributed to cardiac disease. In other words, we cannot speak now of a "cardiac psychosis" and describe a definite clinical entity though, as Riesman points out, there is certainly a syndrome of mental symptoms which is often associated with cardiac pathology. We have observed the course of a chronic psychosis complicated by added mental symptoms, such as affective depression, which it seemed fair to attribute to heart involvement; on the other hand, patients with serious cardiac conditions often in the decompensation stage may present a typical mania or depression, apparently uninfluenced by the somatic disease.

STRECKER, Philadelphia.

ZUR KENNTNISS DER GANGLIO-ZELLULAEREN HIRNGESCHWUELSTE (A CONTRIBUTION TO FURTHER THE KNOWLEDGE OF GANGLIOCELLULAR BRAIN TUMORS). R. HERMANN JAFFE, *Virchows Arch. f. path. Anat.*, Supplement to **227:27** (March) 1920.

The author remarks on the difficulty in differentiating proliferated glia cells from ganglion cells, and on the unreliability of the older communications in regard to this subject. Schminke was the first to describe a typical ganglioneuroma of the brain and Pick, Bielschowsky, Achucarro and Robertson have since described cases of ganglioneuromas made up of more or less typical, often enormous, ganglion cells, nerve fibers and glia elements.

1. Dana, C. L.: Somatic Causes of Psychoneuroses, *J. A. M. A.* **74:1139** (April 24) 1920.

Large cells have also been found in Strümpell-Westphal's pseudosclerosis and in tuberous sclerosis of the brain. Alzheimer described giant glia cells in pseudosclerosis which degenerate without any tendency to form fibers. Many of the cells were almost filled with large nuclei with irregular outlines and projections. In tuberous sclerosis identification of the large cells is not so easy, and Bielschowsky has shown that the picture in tuberous sclerosis is not a unified one, but that both ganglion cells and glia cells must be sharply differentiated in these cases, the ganglion cells appearing only in the cortex and glia cells being greatly in excess. Some authors claim that the difficulty in differentiating the two types lies in the embryonic type of the large cells and is a proof of the congenital character of the process.

Following the consensus of opinion at present, Recklinghausen's neuro-fibromatosis and tuberous sclerosis are one and the same process caused by developmental anomalies in the spongiocytes (principal cells of Schwann's sheath and glia cells), but with different localization. Sclerotic plaques are frequently found in the cortex of Recklinghausen's disease in which large cells are predominant.

The author's case offers no clinical details as the patient was found in a comatose condition, and no previous history could be obtained. Macroscopically the meninges appeared normal; the left temporal region was swollen and the broadened convolutions were hard, leather-like to the touch and on section were pale yellowish-gray, little differentiated from the pure white of the medulla. The posterior portion of the convolution involved in the sclerotic process showed a pigeon-egg sized protuberance to which the pia is closely attached and which is a bright reddish-gray color on section.

Microscopic examination showed that the hardened portion consisted of an enormous development of glia fibers with here and there small wartlike projections containing great numbers of cells. Cells resembling normal glia cells were in the majority in the medullary areas—round nuclei almost without protoplasm. Other cells were seen with larger and paler, sometimes irregular, nuclei. Occasional cells showed much protoplasm, and their nuclei were usually laterally placed. The nervous tissue in these regions was severely injured in all its elements.

The small round tumor body seen in the posterior inferior portion of the affected region showed an entirely different picture and furnished the real subject of the author's article. Enormous cells—from 70 to 90 microns—were seen embedded in a network of fibers. The cells had long, branching processes which formed a network with fibers lying between the cells. Many cells had several nuclei which are near the border of the cell body and bound together by fine chromatin threads. The form and amount of chromatin in the nuclei vary greatly. Sometimes only one nucleus is present, but it is of great size, almost filling the cell. The more normal looking nuclei show a fine chromatin net and one or more well stained round nucleoli.

The author goes into great detail over the fibrillary content of many of these cells, describing many forms of cells with and without dendrites, etc., and concludes that a large number of the cells are ganglionic and that the small tumor is a glioganglio-blastoma of the brain. No Nissl's granula were observed in any of the tumor cells described.

The illustrations lack much in clearness of detail and in convincing power as to the correctness of the author's conclusions.

GURD, Ann Arbor, Mich.

CONTRIBUTION A' L'ETUDE DE L'ANATOMIE PATHOLOGIQUE DU DELIRE AIGU IDIOPATHIQUE (CONTRIBUTION TO THE STUDY OF THE PATHOLOGIC ANATOMY OF ACUTE IDIOPATHIC DELIRIUM). L. REDALIÉ, Schweiz. Arch. f. Neurol. u. Psychiat. 7:35-48, 1920.

In reviewing the literature on this subject, the writer lays particular stress on the work of Ladame, whose contribution was abstracted in the March, 1920, issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, page 324.

The existence of an acute idiopathic delirium has been disputed by numerous writers, who view it not as a morbid entity, but as always symptomatic of some other malady. Redalié, however, believes, as does Ladame, that it is a morbid entity which is quite distinct from that of acute symptomatic delirium which occurs in the course of various infections and encephalitis. Ladame estimates its frequency as 0.25 per cent. of all psychoses.

The case reported in this connection was that of a woman, 43 years of age, who suddenly developed an acute psychosis, characterized by mental confusion, marked motor agitation, sitophobia and progressive general and cardiac weakness, which terminated in death after ten days. The illness followed a violent psychic shock based on the imprisonment of her husband. Necropsy revealed a small endothelioma attached to the dura mater in the region of the cerebellum. The blood vessels of the meninges were markedly injected and the subarachnoid spaces were edematous. The nerve cells of the cortex presented degenerative changes, without pronounced destruction, however; the nuclei were poor in chromatin. The neuroglia cells showed marked proliferation, particularly in the neighborhood of the blood vessels and about the prolongations of the pyramidal cells. There was marked neuronophagia. In the medullary substance, notably about the blood vessels, there was likewise an increase of glia cells. No changes of consequence were noted in the myelin or in the axis cylinders. Changes in the cerebellum were extensive and the description of these is essentially original with this writer. The granular layer was enormously changed by edema, the entire structure being transformed into a system of irregular cavities; the cells themselves contained pyknotic nuclei. Here and there, there seemed to be complete disappearance of the Purkinje cells; the cytoplasm of many stained an intense blue with toluidin, other cells were very pale and without nuclei or nucleoli. A scar, probably the result of an old ependymitis was found in the right lateral ventricle. In addition to these findings, there was epicardial ecchymosis and purulent bronchitis.

The cause and the mechanism by which these changes come about are unknown. Ladame thinks the changes are initially chemical in nature and due to lecithin disintegration; these, he thinks, may well be precipitated by a psychic trauma alone. Redalié thinks that infection has little to do with the disease, basing his belief for this assumption on the fact that there was complete absence of fever, absence of any history of preceding infection, of inflammatory foci in the brain, of hemorrhages or of evidence of transportation of disintegrated myelin; there was, on the other hand, a marked generalization of the process as the horn of Ammon was the only portion of the brain intact. The cause, he thinks, is an auto-intoxication, but he makes no attempt to formulate a more tangible hypothesis further than calling attention to the similarity of some of these findings to those described by Michailois in a case of Asiatic cholera.

The extensive damage done to the cerebellum was quite unexpected as there were no symptoms noted during the life of the patient that would direct attention to this organ. The only finding that might have been attributed to this was the absence of the patellar reflexes; it is possible that incoordination may have been present but if so, it was completely disguised by the extreme agitation of the patient.

WOLTMAN, Rochester, Minn.

LA POLYURIE HYPOPHYSAIRE (HYPOPHYSIAL POLYURIA).

E. SCHULMANN and R. DESOUTTER, *Rev. de méd.* **37**:441 (Sept.) 1920.

Under the name diabetes insipidus, or essential polyuria, one designates a syndrome characterized by an exaggerated and persistent emission of urine without glycosuria or albuminuria, and independent of any renal, cardiac or arterial disease.

The relation of diabetes insipidus and of hypophysial polyuria to each other is unsettled, though the frequent occurrence of anatomic hypophysial lesions in cases of diabetes insipidus is noted, and the author uses the term diabetes insipidus and hypophysial polyuria interchangeably. Is hypophysial polyuria a syndrome of pituitary hyperfunction, of hypofunction or of dysfunction?

Hypophysial polyuria may begin extremely suddenly. In some instances, the patient can recall the day or even the hour when the volume of urine augmented. An injury, a fright, or other strong emotion may, but need not necessarily, mark this day. In other cases, the onset is moderately rapid but spaced over one or two weeks, as a clinical development subsequent to an infection, a head injury, or parturition. Yet other cases are quite insidious and slow in development.

The urinary output in mild cases ranges from 8 to 15 liters in twenty-four hours upward to even 24 liters in that period in other recorded cases. The urine passed during the night period is in excess of that passed during the day. When a normal person and a patient with diabetes insipidus are tested regarding their response to the ingestion of a large amount of fluids, it is found that diuresis begins sooner in the normal. In the insipidus case, the augmentation of diuresis is more tardive but more durable.

The urine in these cases presents two characters. It is normal and it is dilute. The specific gravity is about 1.001 in the severe polyurias of 20 liters and possibly 1.004 to 1.006 in those of only 8 liters. Urinary chemistry does not reveal the presence of any pathologic element, provided there is no concomitant disease condition.

The writer is convinced that (apart from the coexistence of a renal lésion distinct in itself) the kidney in diabetes insipidus is not incapable of reaching a functional concentration of urine. This opinion seems justified since he has demonstrated a concentration of urine in persons with diabetes insipidus (1) in the course of febrile episodes, (2) in the course of partly successful attempts to suppress the ingestion of fluid, (3) under the influence of hypophysial medication, and (4) by adding to the habitual régime a salt, such as sodium chlorid or urea.

The arterial tension is neither exceptionally high nor particularly low and variations in urinary volume do not echo modifications of pressure.

The polyuria does not appear to be a function of blood pressure nor of an urea secretory constant. It is the result of a lowering of the kidney threshold for water. It is an exaggerated permeability of the kidney to water.

DAVIS, New York.

UEBER EINEN FALL VON ENCEPHALOMYELOMENINGITIS
TYPHOSA (A CASE OF ENCEPHALOMYELOMENINGITIS
TYPHOSA). HEINRICH AUGUST MÜLLER, *Deutsch. Ztschr. f. Nervenhe.*
66:168, 1920.

The fact is emphasized that abdominal typhoid fever frequently is associated with severe symptoms attributable to the central nervous system when anatomic lesions are not found of sufficient importance to explain the symptoms. Strümpell especially notes this fact in his textbook.

The author reviews the work of a great number of authors who, however, are far from furnishing a definite picture of the changes in the central nervous system caused by typhoid, and all the phenomena noted are seen in a variety of other diseases.

Probably the majority of observers today are of the opinion that the severe symptoms referable to the central nervous system are due to molecular injury of the nerve cells by the typhoid virus and not by the long continued elevation of temperature. Gerhardt is quoted as describing cases in which a lowering of temperature occurs concomitant with severe brain symptoms, and when the temperature rises these symptoms disappear. Gerloczy, Fränzel and others are cited in favor of the toxic theory.

Many cases are also cited in which a meningitic syndrome was present, but an examination of the spinal fluid showed that a diagnosis of meningitis was incorrect.

On the other hand, many cases of meningitis are reported as occurring with typical abdominal typhoid, but the author's case differs from those already noted by the absence of intestinal localization and the presence of a typhoid meningitis. The diagnosis was made in life by finding typhoid bacilli in the spinal fluid which were placed in culture mediums with positive cultural results.

The clinical history of the author's case was briefly that of a man aged 48 years who became suddenly ill after eating a meat pie. He suffered from nausea and a feeling of heat, but continued in service for ten days. From the ninth day his temperature was taken regularly and reached 39 C. every evening with morning remissions. On the eighteenth day of his illness a careful examination showed pain in the occipital region with tenderness on pressure and much diminished knee jerks. Later he developed diplopia, bladder trouble and paresis of both legs with normal temperature.

There appeared progressive difference in pupils, slow reaction to light, paralysis of the right abducens and right facial nerves, also of the motor fifth and of the twelfth nerve, complete loss of patellar reflexes, etc. The patient's mind was perfectly clear during this period (from the twentieth to the twenty-third day of his illness). Neither typhus nor paratyphus bacilli were found in the blood, but Widal's reaction was positive, 1:1200. The paralytic symptoms increased in intensity up to a certain point, and sensory symptoms were present, but there was considerable variation in the severity of the paralysis up to the thirty-first day of his illness when his mind became unclear, he had difficulty in breathing and finally died.

Necropsy examination revealed small hemorrhages in all the internal organs, intestines included, but none of the ordinary lesions of typhoid in the intestines.

The brain showed some opacity of the pia-arachnoid but no alteration in the cranial nerves or basal vessels. The cortex showed extreme paleness of the white matter with hyperemia of the gray matter.

Microscopic examination showed small cell infiltration around the vessels and some small hemorrhages in the peduncles, in the oculomotorius nuclei, in

the locus ceruleus and in the hypoglossus area. The nerve cells in all these regions were practically unaltered. A few small softenings with large phagocytic cells were seen near the locus ceruleus. The manifestations were most severe in the peduncles and more severe in the medulla than in the pons. The spinal cord showed small cell infiltration about the vessels, severe hemorrhages and degeneration in the lateral columns. The horn cells were unaltered.

The pia arachnoid, particularly of the base of the brain, the medulla and the cord showed infiltration by leukocytes, especially around the vessels. The endothelial lining of the capillaries and the small vessels showed some swelling.

The author goes into the differential diagnosis at length, but probably the greatest value of the article lies in the full bibliography of articles on, and allied to, the subject under discussion.

GURD, Ann Arbor, Mich.

MEASLES: BRAIN COMPLICATIONS. A. L. SKOOG, J. A. M. A. **74**:1697 (June 19) 1920.

The author reports two cases in which both patients had brain complications following measles. One patient had a cerebellar syndrome, and made a complete recovery within six weeks. This case, the author believes, represents one in which the cerebellum and possibly the cerebellar tracts were involved directly from the virus of measles or its toxin. The second case was a complication involving the meninges and the cerebrum. Originally, this was probably a meningitis, appearing a few days after the onset of the measles, with some secondary septic organism as the cause.

From a neurologic review of the two cases reported and an analysis of the literature, the inference is drawn that measles are caused by some virus or organism as yet unseen and uncultivated; further, that the complications and sequelae involving the brain, spinal cord and peripheral nerves are uncommon. Still such cases do occur and may be divided into three groups. The first group would include a minor number of cases in which measles would be merely incidental in their relationship to the complication. The second group, by far the largest, would include complications due to a secondary invading organism. The third group, being a less certain one, would include conditions in which the exact etiology speaks for a direct invasion of the brain by the possible organism causing measles, or its toxin.

The prognosis depends entirely on the severity and location of the pathologic lesions in the brain. Very little is given relative to the treatment of these complications. In a final summary the author states that possibly a number of cases of obscure neurologic disorders, including those with a neurosthenic syndrome, might have had their origin in troubles involving the brain or meninges complicating measles or allied contagious diseases.

ADSON, Rochester, Minn.

THE IMPORTANCE OF VAGAL AND SPLANCHNIC AFFERENT IMPULSES ON THE ONSET AND COURSE OF TETANIA PARATHYROPRIVA. W. L. PALMER, Am. J. Physiol. **52**:581 (July) 1920.

This study was suggested by the frequent clinical association noted between tetany and gastro-intestinal disorder. Two methods of procedure were followed in these experiments. In the first, the effects of double vagotomy and splanchnectomy were studied on the course of tetany developing in a series of

sixteen dogs, following complete thyroparathyroidectomy. A three stage technic was followed in this group, that is, at the first operation the left splanchnic was destroyed, thoracically, and both vagi were cut; following this, at weekly intervals, the right splanchnic nerve and celiac ganglion were removed, and complete thyroparathyroidectomy was performed. In the second method of procedure, the effect of gastro-intestinal irritation (from 2 to 4 minims of croton oil daily) was determined on the tetany occurring in a series of fourteen thyroparathyroidectomized dogs.

The results obtained in this study were entirely negative. In the first series, 64 per cent. of the subjects developed tetany, a figure closely approximating the usual incidence of tetany in thyroparathyroidectomized animals, without vagotomy or splanchnectomy. In the second series, tetany developed in 78 per cent., but the disturbance was not so severe or prolonged as in the first group. It is of interest to note that the frequent vomiting observed by Carlson and Jacobson occurred in two animals of the first series, thus indicating a central origin for the emesis of tetany. The author concludes that neither vagotomy and splanchnectomy nor artificial gastro-intestinal irritation have any influence on the onset and course of tetania parathyropriva, and suggests the possibility of some chemical relationship in the association of gastro-intestinal disorder and tetany.

RAPHAEL, Kalamazoo, Mich.

LA SACRALISATION DE LA V^e LOMBAIRE (THE SACRALIZATION OF THE FIFTH LUMBAR VERTEBRA). L. DELHERN and THOYER-ROZAT, *Bull. méd.* **36**:6 (Jan. 1) 1921.

The authors call attention to an anatomic abnormality consisting of fusion, complete or incomplete, of the fifth lumbar vertebra, with the sacrum. This varies in degree from a relative enlargement of one or both transverse processes to a condition in which the body of the vertebra itself, owing to the great hypertrophy of a transverse process, is directly continuous with the sacrum and iliac bone.

The condition occasions pain symptoms explicable on a basis of root irritation, compression of surrounding tissue, ligamentous strain or possibly on a basis of an "arthritis" of the newly formed articulation between the hypertrophied process and sacrum or ilium. The diagnosis rests on radiographic discovery of the anomaly. Concomitant lesions must not be unsought, however. There is, especially, a tendency for the condition to be associated with spina bifida.

The writer believes that galvanism of the region, thermic penetration and particularly radiotherapy, are effective as treatment. In France, Nové-Josserand and Manclaire have practiced surgical removal of the enlarged processes.

DAVIS, New York.

BRAIN TUMOR AND TRAUMA. HUBSCHMANN, *Deutsch. Ztschr. f. Nervenheilk.* **66**:1 (May 26) 1920.

Hubschmann directs attention to the opportunity afforded by the war for the study of the relationship between head injuries and brain tumor. After giving brief histories and necropsy records in two cases of brain tumor in which a history of trauma was given, he reviews the findings of a number of other writers and gives the results in 107 brain tumor cases in which the

histories were accessible. In 17 per cent. of these cases there was a history of trauma, but in six of them either the injury was slight or head symptoms had been noted before the injury. In the remaining 11 per cent. there were a number in which the relationship between accident and tumor was doubtful. For instance, a 48-year-old man fell downstairs three months before his death. Three weeks later definite tumor symptoms were noted. Section showed a glioma. The accident here was doubtless due to an attack of dizziness caused by the already existing brain tumor. In only three cases were signs of a previous injury to be discovered at necropsy. G. B. Gruber is quoted to the effect that the latter found no case in his war material in which a blastoma could be said to be directly due to trauma, but the possibility of quickened growth, with manifestations of a heretofore existing occult glioma, is admitted.

INMAN, San Francisco.

A STUDY OF FORCED RESPIRATION; EXPERIMENTAL PRODUCTION OF TETANY. S. B. GRANT and A. GOLDMAN, *Am. J. Physiol.* **52**:209 (June) 1920.

Grant and Goldman report the consistent appearance of all the essential symptoms of tetany as a result of forced respiration in each of twenty-four experiments on two human subjects.

The subjects were required to breathe as deeply as possible, at the rate of fourteen inhalations a minute, while in the supine position, until symptoms of tetany developed, usually in from fifteen to sixty minutes. Blood and urine examinations were made, and the alveolar carbon dioxid tension measured, before and after each experiment. Diagnosis of tetany was based on the presence of carpopedal spasm, Chvostek's sign, Trousseau's sign, Erb's sign of increased electrical irritability and, in one instance, tetanic convulsion.

In addition to symptoms of tetany, there was found, uniformly, a fall of alveolar carbon dioxid tension which resulted in a reduction in hydrogen-ion concentration of the blood, a reduction of the carbon dioxid capacity of plasma, a change of urinary reaction to the alkaline side, a decreased excretion of ammonia and a slightly increased serum calcium content. The last finding is of especial interest as it stands in direct contrast to observations in clinical tetany made by other workers, notably by MacCallum and Voegtlin, MacCallum and Vogel, and Howland and Marriott.

In concluding, the authors suggest that tetany be regarded as a syndrome which probably may be caused by any condition tending to heighten the irritability of the peripheral nerves as, for example, in their study, the systemic alkalosis produced through forced respiration.

RAPHAEL, Kalamazoo, Mich.

NERVE SUTURE. EDWIN A. MILLER, M.D., *Arch. Surg.* **2**:167 (Jan.) 1921.

This experimental study was undertaken to ascertain the tensile strength of the suture line when enough of the nerve has been removed to require fixation in marked flexion of the limb. The conclusions are:

1. In dogs which show individual differences in rapidity of repair, as human beings do, the tensile strength of a suture line in the sciatic nerve or its branches is practically as great at the end of the third week as at the end of the fourth or fifth week.

2. The strength of the suture line, especially after the second week, is almost directly proportional to the diameter of the nerve.

3. The epineural sutures of fine catgut or silk play little, if any, part in the strength of the suture line, after the second week.

4. Long defects of nerves may be overcome by mobilization of the segments and posture, an end to end suture being performed. The suture line is apparently firm enough after three weeks to begin gradual straightening of the flexed forearm only. Clinically, after operation on the sciatic nerve, it would seem best to wait six or eight weeks after suture before extending the leg of flexion to complete an end to end suture. In case of the median and ulnar, extension of the forearm should not be attempted until after weeks of flexion.

RODMAN, Philadelphia.

LA ENCEFALITIS LETHARGICA EN ESPAÑA (LETHARGIC ENCEPHALITIS IN SPAIN). G. R. LAFORA, Arch. de Neurobiol. 1: 209 (June) 1920.

This is a clinical and anatomopathologic study. Lafora summarizes the clinical and pathologic findings published, beginning with Economo and extending to the date of this article. He then presents several cases similar in clinical character and histopathology to those mentioned and goes into the theories of causation, etc. He offers nothing new, but he stresses the difference in the histopathologic picture of lethargic encephalitis (a more or less chronic inflammatory process with infiltration by lymphocytes and their derivatives, plasma cells, etc.) and that of encephalitis occurring in influenza in which the lesions are purulent and hemorrhagic in type. He also notes the resemblance of the lesions in lethargic encephalitis to other brain lesions of parasitic origin, such as rabies, syphilis and sleeping sickness. He makes a point which he considers of importance, that is that every tissue has a characteristic reaction in relation to different germ groups, the brain for instance reacting to microbes (meningococcus, diplococcus, staphylococcus, etc.) by polymorphonuclears thus giving rise to acute meningitis or to abscesses in the substance of the brain, on the contrary reacting to parasites by lymphocytic focal or diffuse infiltrations of the blood vessels and meninges, Koch's bacillus alone furnishing a parasitic reaction.

GURD, Ann Arbor, Mich.

POLYNEURITIS FOLLOWING INJURIES. FRIEDRICH LEPPMANN, Ztschr. f. die ges. Neurol. u. Psychiat. 49:198 (July 11) 1919.

Leppmann takes up the question as to whether widespread nerve inflammation as a sequence of trauma can occur in the absence of external injury and suppuration. In addition to several doubtful cases cited from the literature, he gives the history of one case observed by him in which the element of accident compensation came into question. However, the possibility of a poliomyelitis or of the effect of overindulgence in alcohol could not be excluded. Numerous brief case histories are cited in which a more or less widespread neuritis followed infected wounds.

In conclusion Leppmann states that there is no evidence in proof of a widespread neuritis resulting from uninfected wounds, and that a polyneuritis following trauma without tissue laceration or infection will be found to be due to some other cause, such as diphtheria.

INMAN, San Francisco.

A PLETHYSMOGRAPHIC STUDY OF SHOCK AND STAMMERING IN A TREPHINED STAMMERER. S. D. ROBBINS, *Am. J. Physiol.* **52**:168 (May) 1920.

The subject of this plethysmographic study was a man, aged 45, who, as a result of operative interference, fifteen years previously, following a bullet wound of the head, was left with a slightly depressed trephine opening, about 2.5 cm. in diameter, in the right frontotemporal region, 5 cm. from the mid-line. There was a history of stammering since early childhood, following fright but, at the time of the experiment, speech was practically without defect when the subject was alone.

On the basis of this study, the author reports marked increase in brain volume, in association with stammering, as compared with the lesser increase determined in the course of normal speech, thus corroborating Bluemel's hypothesis. Robbins also noted increased brain volume in association with strong emotional ("shock") reaction and mental and physical work, bearing out earlier work by other observers, notably by Berger, Mosso, Shepard and Weber.

RAPHAEL, Kalamazoo, Mich.

VARIATION DU TAUX DE L'UREE DANS LE LIQUIDE CEPHALO-RACHIDIEN PRELEVE AU MOMENT ET EN DEHORS DES CRISES CONVULSIVES EPILEPTIQUES ET HYSTERIQUES (VARIATIONS IN THE UREA CONTENT OF THE CEREBRO-SPINAL FLUID OF EPILEPTIC AND HYSTERIC PATIENTS, TAKEN DURING CONVULSIVE SEIZURES AND AT OTHER TIMES). GASTON LAURÈS and EMILE GASCARD, *Presse méd.* **28**:396 (June 16) 1920.

"Whatever the urea content may be at other times, it is lessened during the hysterical and increased during the epileptic convulsion." To support this thesis, the findings in six frankly epileptic and six hysterical cases are cited; also in two doubtful cases, which yielded results of epileptic type and later developed unquestioned clinical epilepsy. Uremic and syphilitic convulsions, if definitely epileptiform, were not positively differentiated from idiopathic grand mal, and the fluids of such cases conformed to the epileptic type.

Lumbar puncture was performed during a postconvulsive phase, and again four days later, in each case studied. The following figures represent grams of urea in 1,000 c.c. of cerebrospinal fluid: average epileptic content during convulsion, 0.55; otherwise, 0.33; during hysterical convulsion, 0.28; otherwise, 0.47.

HUDDLESON, New York.

DISEASE OF THE PERIPHERAL NERVES IN THE WAR. ERWIN WEXBERG, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **49**:198 (July 11) 1919.

In this article of seventy-one pages, the author presents case histories and discusses the probable causes of the peripheral nerve disturbances occurring in the Maria Theresa Schossl in Vienna. The cases fell into five groups. In nine of the fifteen cases in the idiopathic polyneuritis group, exposure to cold had been given as the causative factor. However, in only one case did this cause alone seem to be culpable. In three other patients there had been associated nonspecific gastro-intestinal disturbances, while the remainder had suffered from undernourishment, which was assumed to have reduced their resistance.

INMAN, San Francisco.

SOBRE LA COMPOSICION Y LAS PROPIEDADES DEL LIQUIDO
CEFALORAQUIDEO EN LA ENCEFALITIS LETARGICA (A
STUDY OF THE COMPOSITION AND PROPERTIES OF THE
CEREBROSPINAL FLUID IN LETHARGIC ENCEPHALITIS).
W. MESTREZAT and BELARMINO RODRIGUEZ, Arch. de Neurobiol. **1**:227
(June) 1920.

Four cases are studied and the authors conclude that in lethargic encephalitis, with the exception of hyperalbuminosis and hypercytosis, the cerebrospinal fluid is essentially and generally normal and its chemical, cytologic and serologic examination offers an important diagnostic method in this disease, which is so variable in its clinical manifestations.

GURD, Ann Arbor, Mich.

Society Transactions

THE CHICAGO OPHTHALMOLOGICAL AND THE CHICAGO NEUROLOGICAL SOCIETIES

Joint Meeting, Dec. 16, 1920

ALFRED N. MURRAY, M.D., *President of the Chicago Ophthalmological
Society, in the Chair*

A CASE OF POLIENCEPHALITIS SUPERIOR AND INFERIOR. DR. G. B. HASSIN.

This article is published in full in this issue, p. 552.

DISCUSSION

DR. HIRAM J. SMITH said that the cause of ocular paralysis may be orbital or intracranial. The intracranial may be considered as supranuclear, nuclear fascicular, or superficial. In extensive progressive ophthalmoplegia the lesion is nearly always nuclear. Supranuclear lesions, that is, of cortex association centers and intracerebral tracts, cause conjugate paralyzes, seldom isolated paralysis, with the exception of ptosis. In this type of conjugate paralysis, the eyes usually are able to turn toward the affected side of the brain, but not toward the opposite, that is, "look to the lesion," as contrasted with conjugate paralysis of pontile origin, in which the eyes might turn from the lesion.

Bilateral ophthalmoplegia is not necessarily due to bilateral involvement of the nuclei. Fibers from nuclei of a given side pass to the nuclei of the opposite side so that a lesion of the right third nucleus may cause a disturbance of the muscles of the opposite side, as ptosis. The affection of the opposite side, in a case under observation, cleared up in forty-eight hours, probably through compensatory action of the unaffected nucleus, as the paralysis on the side of the lesion persisted.

In progressive nuclear involvement, one would naturally expect the adjacent nuclei to be affected at the same time, or in succession; and this happened. The third and fourth, or sixth and seventh, were involved together, as well as adjacent nuclei of other cranial nerves.

The diagnosis of the cause of ocular paralysis might be suggested by the type and extent of the trouble. In nuclear paralysis the underlying affection would be cleared up, usually through the finding of other manifestations than the ocular. The characteristics of multiple sclerosis were readily perceived. In bulbar paralysis the early involvement of hypoglossus and glossopharyngeus was encountered. In myasthenia gravis double ptosis was seen early, but rapid fatigue of the muscles of the head and neck, especially muscles of mastication, was characteristic. Nevertheless, many obscure clinical pictures presented themselves.

DR. PETER BASSOE thought it would be profitable if Dr. Hassin would emphasize the distinction between poliencephalitis superior and inferior caused by other infections and the purely degenerative affections of the same regions. A similar problem had been worked out in the case of the spinal cord. For a

long time all conditions were called myelitis without sufficient distinction between inflammation, degeneration and vascular lesions.

DR. H. DOUGLAS SINGER stated that, according to the statement of the essayist, the spinal fluid was absorbed apparently both through the arachnoid villi and through the choroid plexus. He wondered what the source of the spinal fluid was and whether it was true that the fluid was absorbed at both sides of the brain. The usual view was that the choroid plexus acted as a gland to create the fluid, but Dr. Hassin apparently had an altogether different view.

DR. MICHAEL GOLDENBURG said that he was under the impression that the spinal fluid was secreted by the choroid plexus, and that the epithelium covering it was merely a filter.

DR. HUGH T. PATRICK asked how the fat and epithelial cells in the choroid plexus got there from the spinal fluid, and whether anybody had ever found fat in the spinal fluid in this sort of case.

DR. HASSIN, in closing the discussion, said that he did not intend to consider in detail the important physiologic points suggested by the pathologic studies of a remarkable case. He merely wished to demonstrate their probable significance. The masses of lipoid substances in the gray matter of the mid-brain and medulla were striking, but nobody had ever noted their presence in the subarachnoid space and the choroid plexus. Evidently, fatlike substances had not been looked for in these regions or proper methods were not used. In fact, few histopathologic studies of so-called hemorrhagic superior poli-encephalitis had been recorded, the authors contenting themselves with repeating what Wernicke had said. Schroeder and Spielmeyer were the first to point out that Wernicke's poli-encephalitis was not an encephalitis at all. In his (Dr. Hassin's) opinion the only true superior poli-encephalitis was represented by epidemic (lethargic) encephalitis in which the inflammatory phenomena were principally, though not exclusively, confined to the midbrain. In Wernicke's type the morbid process had the same localization, but was of a degenerative, and not of inflammatory, character.

As to the probable function of the choroid plexus, he wished to state that, according to some authors, the cerebrospinal fluid originates partly in the brain, partly in the choroid plexus. The abundance of fat in both these structures indicated that their contents were wholly derived from the brain tissues. In the case under discussion, these contents were lipid substances; in cerebral hemorrhages they would be blood pigment and so forth. The choroid plexus, therefore, was to be looked on as a filter for the cerebrospinal fluid, which it rendered passable through the various channels of absorption. Generally speaking, the study of pathologic brain conditions might help to solve problems which so far defied the efforts of the ablest experimental workers.

THE PUPIL IN HEALTH. DR. E. V. L. BROWN.

Dr. Brown stated that, according to Salzmann, the pupil in health had a diameter of approximately 4 mm. The consensual reaction depended on the stimulation of the rods and cones in the relatively small area of the macula. The stimulus was then carried by the optic nerve to the chiasm, where partial decussation took place, thence via the tractus opticus with the pupillary fibers lying dorsolateral to the corpora quadrigemina, and finally to the nucleus of the oculomotor nerve, which functioned as the pupil nucleus as well. Through

the fibers which crossed over from the right to the left side, therefore, any stimulus of the right macula went to the left pupil, centered as well as to the right and was then sent down the left oculomotor to the sphincter of the iris on each side, the left pupil narrowing at the same time the right did. This test was of the greatest value in establishing the functional integrity of the most vital part of any injured eye. Many a patient who had just suffered a severe accident to a considerable portion of the front of his eye could easily and quickly and honestly be told that the eye was not lost, even when the cornea was cut, the anterior chamber full of blood, the iris prolapsed or the lens dislocated, for the pupil of the fellow eye narrowed when light was thrown into the injured eye. One of the uncanny things about the consensual pupil reaction was the fact that in rare instances of disease of the cortex, such as tumor of the occipital lobe, etc., one eye actually did not see, or rather the cortex of neither side saw, yet the pupil motor stimulus was sent up the one optic nerve, across to the other side and down that oculomotor nerve, and the pupil of the opposite side narrowed as perfectly as if light and color and form perception were perfect.

Widening of the pupil takes place through irritation of the sympathetic nerve. The pupil widening fibers leave the spinal cord at the level of the upper two dorsal and the lower cervical segments. Fibers from the upper thoracic ganglion and the anterior branch of the ansa Vieussensii go to the inferior cervical ganglion, and out of it into the cervical ganglion via the cervical sympathetic. Here there is union with the hypoglossal. The carotid branches are then given off and the pupillodilator fibers proceed in the skull to the Gasserian ganglion and unite with the first branch of the trigeminus. So united they proceed to the eye via two long ciliary nerves to the dilator sheet of muscle in the back layers of the iris. They do not pass through the ciliary ganglion at all.

Any irritation of the cervical sympathetic can, therefore, produce dilatation of the pupil. Furthermore, the irritation or stimulation of any sensory nerve may produce a dilatation of the pupil. The path here is to the cerebral cortex, the oculomotor nucleus and to the iris via the third nerve, ciliary ganglion and short ciliary nerves to the sphincter pupillae, which relaxes and allows the dilator to work unopposed. Furthermore, the pupil widens on any psychic stimulus, and volitional impulse and any vivid mental concept.

DISCUSSION

DR H. DOUGLAS SINGER stated he often found recorded: "pupils sluggish to light," and he had never been able to satisfy himself as to what most people meant by sluggishness. Did it mean that the reaction was slow or that the degree of contraction was diminished?

In his opinion the fibers that conveyed the stimulus for the light reflex left the optic tract before it reached the pulvinar. They apparently left in the region of the thalamus and traveled along the inner side of the thalamus. This seemed to be proved by two cases of tumors seen many years ago, involving the back part of the third ventricle and damaging the optic thalamus on both sides in both of which there had been Argyll Robertson pupils.

DR. HUGH T. PATRICK stated that the dictum of Uthoff that even if there was more illumination of the pupil on one side, the pupils remained equal, was wrong.

Another curiosity could be referred to as a normal pupil: It was known that occasionally a person could voluntarily dilate his pupil by picturing to himself some peculiarly horrible scene, generally from his own experience.

DR. RALPH C. HAMILL said that in testing the pupils, especially of colored men with dark irides, it was difficult to tell whether there was a light reflex or not. Dr. Brown had mentioned the fact that the near-sighted person has small pupils, and he wondered whether in some persons where the pupil was under more or less spasm small changes of size would be visible. Also, in testing the pupils of a great many men in a short space of time, as was done with some of the men in the training camps, it was observed that there were certain kinds of pupils that corresponded to the degree of pigmentation of the iris.

DR. I. LEON MEYERS thought that the influence of the sympathetic nervous system, and especially that which was noted in emotional disturbances, fright, etc., was not the only one that brought about dilatation of the pupil. It had been noted many years ago that in stimulating the cortex of an animal while it was completely anesthetized and the stimulation was strong enough to produce epileptiform fits, the pupils would promptly dilate. This had no connection with stimulation of the cortex when it produced conjugate deviation of the head and eyes.

DR. ROBERT VON DER HEYDT stated that as to light and dark irides; there were at least two reasons why eyes with dark irides did not respond to light as well as those with lighter colored irides. One was a sluggishness in response on account of the weight of the added pigmentation in dark irides. Then, light would penetrate a light colored iris more readily on account of its greater transparency, and the retina would receive more stimulation for that reason.

DR. H. W. WOODRUFF spoke of the statement made in the standard textbooks on ophthalmology that "inequality of the pupils was always pathological." Reference had already been made to the larger pupil in myopia. This also held when one pupil was myopic and one hyperatrophic, namely, in anisometropia. In such a case one pupil was distinctly larger than the other. When he first began the practice of ophthalmology he did not know this and supposed a patient with inequality in the pupils must have a serious nerve lesion. For this reason, in examining these cases, the refraction should be known.

DR. CHARLES P. SMALL said that the differences in the reactions in the normal pupil were illustrated in a case seen recently. The patient was a man in perfect health, with all laboratory examinations negative, who was refused an increase in life insurance because he was said to have an Argyll Robertson pupil. The pupils were widely dilated and almost immobile, but they did react sluggishly when carefully examined. He did not know why he had such feeble reaction, and wished some of the neurologists would explain it to him.

DR. C. W. HAWLEY was reminded of a case similar to Dr. Small's which he had reported. His patient had widely dilated pupils all her life without pupillary reaction. Suddenly the left pupil was contracted to the usual size and developed reaction. She came to have the pupil dilated to look like the other.

As to one pupil dilating more when it was receiving more light than the other, he had seen a similar case within two or three months. During the examination a friend of the patient asked why one pupil was dilated more than the other, and he thought it might be because that eye was receiving more

light than the other. On turning the patient around he obtained the opposite effect and proved that this theory was correct.

Dr. BROWN, in closing the discussion, in reply to Dr. Singer, said he had always understood sluggishness to refer to the rate of reaction rather than the degree. He was glad to hear Dr. Patrick emphasize the fact that when more light enters one eye than the other the pupil of the first eye is narrower than that of the other, thus proving that the direct reaction is greater than the indirect or consensual reaction.

PHILADELPHIA NEUROLOGICAL SOCIETY

Jan. 28, 1921

CHARLES S. POTTS, M.D., *President pro tem.*

A CASE OF PARALYSIS WITH CONTRACTURE OF PHYSIO-PATHIC ORIGIN. Presented by DR. A. J. OSTHEIMER.

An ex-service man, now 20 years of age, fell into a shell hole while carrying a wounded soldier back from the line on July 18, 1918. The result, according to a report from the Adjutant-General's office, was a simple fracture of the body of the left scapula, with dislocation of the left humerus, causing injury to the third, fourth and fifth cervical nerves. A roentgenogram made at the time of discharge, April 9, 1920, showed separation of the glenoid cavity, with some atrophy of the infraspinatus, supraspinatus and deltoid.

When examined, June 29, 1920, there was a good deal of dragging pain in the left shoulder, along the trapezius and in the hand, which at times became somewhat contracted in a flexed position. Both hands and feet were of a somewhat dusky hue, mottled and showed profuse perspiration. He was unable to raise the left arm to the horizontal. There was involvement of the left trapezius, levator scapulae, rhomboidei and to a much lesser extent of the serratus magnus and supraspinatus. As these muscles are supplied chiefly by the third, fourth and fifth cervical nerves, there seemed to have been a rupture of the cervical plexus. The left grip was 35 on the dynamometer, the right 85; otherwise there was no evidence of organic trouble.

A roentgenogram made on July 9, 1920, showed no abnormality. About this time the patient began gradually to develop marked contracture of the left forearm and of the hand on the forearm with paralysis, together with the thermic, trophic and vasomotor symptoms which accompany the reflex disorders, as Babinski and Froment have named them, or the dynamoneuropathic disorders under which name the late E. E. Southard has classified them. All the typical signs of the "main figée," as first described by Meige, were present. The vasomotor disturbances were marked, confined to the hand and did not correspond to any innervation area. The right hand also was cold and showed slight global atrophy, while the skin was of salmon color, wet and mottled. The mechanical excitability of the small muscles of the hand, particularly of the thenar and hypothenar eminences, was increased, as well as the electrical excitability of the same muscles. All the other muscles showed normal electrical reactions. The tendon reflexes were practically normal. The paralysis was almost complete. There were no sensory disturbances.

This case is of interest because the symptoms and signs are different from those that occur in frank lesions of the nerves, as well as from those that are purely pithiatic. The thermic, trophic and vasomotor signs, as well as the resistance to all forms of treatment, makes this condition a distinct entity. The recent splitting of functional cases into the psychopathic and the physiopathic has been well worked out, principally by Babinski and his associates of the French school. Hurst and other English neurologists have held that all of these symptoms are simply consequent on disuse following pithiatic paralysis and contracture. By chloroforming a patient, Babinski has shown that when all the other reflexes were stilled, certain reflexes that were entirely concealed in the waking state were brought out; yet at the same time consciousness in the usual sense of that term had vanished. Was not this proof of a new type of functional disease nonpsychic in nature, but of almost equally complex nature? The chloroform apparently suspended the operation of numerous neurons that have to do with the down flow of cerebral inhibitions and allows phenomena to appear in certain reflex arcs that argue an excess of activity, for example an ankle-clonus or a patellar clonus. This seemed to prove the existence of a disorder of a reflex or physiopathic nature below the level of the psyche and below the theater of operations of hysteria.

The most complete recent review of this subject has been made by A. Pitres and A. Laffaille: "Sur les paralysies globales molles fonctionnelles de la main consecutive à des blessures de guerre," in the *Revue de médecine*, 1920, Nos. 5 and 7.

DISCUSSION

DR. WILLIAM G. SPILLER said this subject is important and has caused much discussion. Roussy in his paper read before the American Neurological Association last spring and in his moving pictures showed that the lesions of "reflex" contracture and paralysis are dependent on functional disturbance. He showed in moving pictures that when a paralyzed hand is moved synchronously with the sound hand, although at first the movement may be slight, the paralyzed hand slowly acquires power, and with the return of voluntary motion the vasomotor and trophic symptoms disappear. The functional character of the paralysis is demonstrated by the improvement under the treatment employed. The hysterical palsy thus seems to condition the vasomotor and trophic disturbances which in reality are organic but secondary to functional disturbances.

DR. T. H. WEISENBURG said that he had seen a number of cases similar to the one reported by Dr. Ostheimer, and he had no doubt that this case was purely functional. In a similar case, which had come under his observation in Plattsburg, by bullying the patient, in about five minutes he was able to extend his fingers whereas previously they were flexed. Subsequently this man was cured. One of the interesting points about all of these functional cases is the rapid disappearance of vasomotor phenomena not only in the case as described by Dr. Ostheimer, but in all cases of hysterical weakness and contracture.

DR. D. NATHAN said that abroad he had seen many cases very much like this in which there were contracture of the biceps tendon and atrophy and contracture of the hand. These cases were often associated with unilateral sweating and other vasomotor phenomena, and he classed them all as hysterical, and cured most of the patients in a few hours or days.

SYPHILIS OF THE EIGHTH NERVE. Presented by DR. J. HENDRIE LLOYD.

Dr. Lloyd based his paper on a series of cases observed in his hospital service. Sudden or rapid deafness, with tinnitus, coming on usually early in the secondary stage and ending in incurable loss of hearing, was the chief characteristic. In some cases there was a cranial polyneuritis, the seventh, fifth, third and second nerves being involved in about the order named. Severe headache with high lymphocytosis pointed to an acute basilar syphilitic meningitis as the cause. An opportunity for postmortem study occurred in one case, and the microscope recorded extensive destruction of the eighth nerve. The paper will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. N. W. WINKELMAN said that he had not yet completed the pathologic examination of the specimen referred to by Dr. Lloyd. The nerve was rather peculiar on both sides. It showed practically complete degeneration, only a few waxy axis cylinders running through. The neuroglia tissue was laid down in columns showing degeneration had taken place slowly. There were some concentrically arranged bodies that were probably myelin bodies in the hematoxylin-eosin and phosphotungstic acid stain, also some infiltrating mononuclear cells very hard to distinguish from the resting stage of the glia cells.

DR. T. H. WEISENBURG said that he did not agree with Dr. Lloyd that arsenic preparations had injurious effects on the auditory nerve or on any portion of the nervous system, and in his opinion they were distinctly the best antisymphilitic drugs we possessed. In the Dermatological Laboratories in Philadelphia the weekly output is over 10,000 tubes, and this laboratory is only one among many. Arsenic has been used for about fifteen years, a sufficient time for us to come to a conclusion regarding its influence on the nervous system, and up to the present there is no evidence of its deleterious effect.

DR. WILLIAM G. SPILLER said that often there is meningitis in tabes, though sometimes slight, but it is not proper to attribute the degeneration of the posterior roots to the meningitis. Syphilis may by its toxic properties directly affect the posterior roots. He felt that sometimes too sharp localizations are made by the Bárány tests, because only one group of fibers in the acoustic nerve seems from the symptoms to be diseased, and the conclusion is formed that the lesion must therefore be where the fibers of this nerve having different functions have separated. This conclusion is unreliable. If one examines the lumbar region in a tabetic cord when death has occurred from some other disease, he will find the degeneration of the posterior columns corresponds to the embryologic picture afforded by the medullation of these columns at a definite period. The syphilitic toxin has attacked the whole of the posterior root, but only certain fibers in this root corresponding to their period of embryological "ripening" have succumbed, although later other fibers in the root would also have degenerated.

There are many examples of the selective action of poisons or pressure on certain groups of fibers in a bundle of nerve fibers. The papulomacular bundle is especially liable to degeneration from alcohol, lead or nicotin, also from pressure on the optic nerve. It alone of the optic nerve fibers may be affected in some cases of multiple sclerosis. Diphtheria may select the branch of the oculomotor nerve to the ciliary muscle without affecting the remainder of

the oculomotor nerve. Lead may select the posterior interosseous fibers from the musculospiral nerve. Therefore, because only the cochlear fibers or the vestibular fibers of the acoustic nerve have caused symptoms, it may be a mistake to assert that the lesion can by no means be within or on the acoustic nerve but must be within the brain stem where the tracts are widely separated. It may be at such a part but it need not be. Gowers has shown that clinically all of the fibers of the acoustic nerve are not invariably equally affected in tabes.

DR. J. HENDRIE LLOYD, in closing, said that in the main he agreed that arsphenamin is given so much with so little bad effects, that it is not fair to ascribe to this drug injurious effects on the auditory nerve. Nevertheless, there may be a weak spot in that argument, for it is the exceptional case that we must bear in mind. He was averse to making sensational statements, but he thought we ought to know the truth.

With reference to the Bärány tests, as he said in his paper, we should be slow in accepting some of the extreme claims made for their localizing value. They apparently have a general value in these cases of peripheral, or eighth nerve lesions, as indicating that some at least of the functions of the vestibular nerve are affected. The microscopic examination shows extensive destruction of the eighth nerve, and to that extent the Bärány tests were accurate in their results. This opportunity to examine the eighth nerve in acute syphilis was a rare one.

A CASE OF RAYNAUD'S DISEASE MUCH IMPROVED BY BRACHIAL SYMPATHECTOMY. Presented by DRs. GEORGE MULLER and GEORGE WILSON.

The case shown was that of a man 72 years of age who had had symptoms and signs of Raynaud's disease in both hands for two winters. Following bilateral brachial sympathectomy there was marked improvement in the vasomotor phenomena in the hands.

The case will be presented in full at a later date.

FIVE CASES OF PITUITARY DISEASE. Presented by DRs. T. H. WEISENBURG, C. A. PATTEN and F. AHLFELDT.

CASE 1.—*History*.—Z. S. H., a white woman, aged 35, whose family history was negative, except for the possibility of syphilis, had a normal birth. She was fat as a baby and delayed walking until she was 4 years old; she did not talk until she was 5. She had a persistent low mentality. The menses were established at 11 years; she had had nine pregnancies with five miscarriages and four stillbirths. An ankle was fractured ten years ago. She has had tremors of the left arm since being struck by her husband four years ago. Her hair became gray at the age of 11.

Physical Examination.—She had disproportionately long extremities; fat, small, pudgy hands; segmental distribution of the body fat, and a low hairline on the forehead. She had an adrenal "white line"; normal blood pressure, and a positive oculocardiac reflex. There were reaction to epinephrin with lowering of pulse (2 beats), increase of blood pressure (10 mm.), and a leukocytic increase from 8,600 to 10,800, a relative lymphocytosis and eosinophilia. She reacted to atropin with a rise of 20 beats in the pulse rate. The

sella turcica was distorted. She had a markedly increased sugar tolerance, and the blood Wassermann reaction was positive.

This case is interpreted as persistent hypofunction of the pituitary gland. In addition, the patient had a neurosis and was syphilitic.

CASE 2.—History.—M. F. was a white woman, aged 40. There had been endocrine disturbances in various members of the family: giantism, obesity, dwarfism, bed-wetting, diabetes and gastro-intestinal disorders. At the age of 5 she changed from a bright and active child to a dull and inactive one. Secondary sexual characteristics appeared at the age of 5, and a sudden increase of growth and weight at 9 coincident with the establishment of the menses. Bed-wetting had been present all her life. Two illegitimate pregnancies followed the awakening of sexual desires at the age of 18. Surgical menopause occurred at 26, with convulsive seizures occurring at intervals since, and coming on about the time of the usual monthly period.

Physical Examination.—The pupils were irregular. "Vessels in the left eye have a turn toward the nasal side on emerging from the nerve head." The body was disproportionately longer than the extremities. She weighed 330 pounds, with segmental distribution of fat. The hairline was low on the forehead. There were an adrenal "white line," low blood pressure and pulse rate. The oculocardiac reflex was positive. There was a reaction to epinephrin with an increase in the pulse rate (10 beats) and blood pressure (15 mm.); a leukocytic increase of 3,800 cells occurred—the lymphocytes and eosinophils were mainly affected. There was a reaction to atropin with an increase in pulse rate of 20 beats a minute. The sella turcica was enlarged. There were increased sugar tolerance, lessened kidney elimination and a positive blood Wassermann reaction.

This case shows definite pituitary deficiency together with thyroid deficiency and artificial menopause. The patient was also epileptic (?) and syphilitic.

CASE 3.—History.—The case of F. F., a white girl aged 14, was fully reported by Dr. F. H. Leavitt before the Philadelphia Neurological Society on Dec. 19, 1919, and appeared in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, April, 1920, on page 452. It is included in this series for contrast.

Her family history was negative. Her birth was instrumental. She had a probable meningitis at the age of 8 months, marked by retraction of the head, convulsions, incontinence, and blindness (which lasted until the age of 2). Walking and talking were delayed. Her mentality had always been low. At 5 she became very fat and weak with evidence of hydrocephalus and lateral column sclerosis. Secondary sexual characteristics appeared at the age of 7 and menses at 12.

Physical Examination.—Examination revealed: enlarged head (60 cm.); small infantile extremities and a disproportionately longer torso; dry, doughy skin; hypotrichosis, and low hairline on the forehead. Recently her hair had been falling out. There was segmental distribution of fat and lymphoid tissue hyperplasia. The adrenal "white line" was present. There were a positive oculocardiac reflex, markedly increased sugar tolerance and an enlarged sella turcica. Reaction to atropin was strongly positive. Neurologically she showed a lateral column sclerosis and divergent strabismus. The blood Wassermann reaction was positive.

This patient revealed a definite hypopituitarism, probably secondary to the meningitis at 8 months and developing coincidentally with the hydrocephalus and lateral sclerosis. She was also syphilitic.

CASE 4.—*History*.—C. T., a colored boy, aged 15, with a negative family history, was normal up to 6 years of age when the right arm and hand began to grow disproportionately larger. At the age of 12 it was discovered that he had a positive blood Wassermann reaction. Antisyphilitic treatment was continued for one year. At 14, he began having convulsions described as typical petit mal and grand mal attacks. For the past few months these attacks have been more frequent.

Physical Examination.—This revealed: disproportionate length and circumference of the right arm, hand and shoulder and disproportionately short torso. The extremities showed proportionately longer proximal than distal length. The sella turcica was normal. There was a slight tufting of the distal phalanges of the fingers. The upper teeth were widely spaced; the canines were not fang-shaped, but very like the incisors. There were: moderately increased sugar tolerance, high lymphocyte count, low kidney function and increased nonprotein nitrogen in the blood. The Wassermann reaction was weakly positive to negative.

This case offers more difficulty in diagnosis. The evidence may possibly point to a hyperfunction of the anterior lobe in childhood, but it seems to have become arrested. At present he cannot be said to have hypopituitarism on the basis of a slightly increased sugar tolerance, except it be that this is evidence of the beginning of deficient function. It would probably be best to classify the case as one of dyspituitarism. The patient was also epileptic and syphilitic.

CASE 5.—*History*.—I. E., a white boy, aged 16, began developing rapidly at the age of 9. His father and mother were syphilitic and were first cousins. The father contracted syphilis at the age of 20, and the patient was the first born. The mother had been pregnant seven times with two miscarriages. There were three sisters and one brother—all showing endocrine disturbances, mainly in precocious physical and sexual development. The girls began menstruating at the age of 8 or 9 years, and their secondary physical characteristics appeared at the same time; the boy, now 16, had fully developed secondary sexual characteristics, axillary and pubic hair, adult genitalia and deep voice.

The patient had "snuffles" at birth. Walking and talking were delayed. He had had otitis media and interstitial keratitis in infancy and scarlet fever following diphtheria at 11, during which illness he began to grow rapidly and to gain in weight. Bed-wetting began also at this time and has continued since. Pubertal changes occurred at 13, but he has no sexual desires. Adult stature was reached at 13.

Physical Examination.—This revealed: partial loss of vision and impaired hearing; marked obesity (he weighed about 230 pounds), with a segmental distribution of the fat; disproportionately long extremities, especially the sacral portions; thin skin but doughy in consistency and hypotrichosis with low hairline on the forehead; about normal pulse and blood pressure. An "adrenal white line"; negative oculocardiac reflex; markedly increased sugar tolerance; low kidney elimination and positive blood Wassermann reaction; reaction to epinephrin with increase of pulse (18 beats) and increase of blood pressure (20 mm.); leukocytosis of 10,000 to 12,000; a positive reaction to atropin (pulse rate, increasing 22 beats) and pilocarpin (decreased 4 beats). The roentgen ray showed an enlarged sella turcica and the convolitional markings of internal hydrocephalus.

This case evidently is one of dyspituitarism; the condition now is that of hypofunction, but evidence points to an earlier hyperactivity, at least of the pars anterior. He also has internal hydrocephalus, optic atrophy, bilateral impairment of hearing, enuresis and syphilis.

Remarks.—A positive blood Wassermann reaction is now, or has been, present in all five cases. The rôle which syphilis has played in the etiology of the dysfunction of the gland is perhaps not clear, yet the coincidence is striking. In Case 5 there is no doubt about the heredity of the disease and Case 4 is quite certainly congenital; in Cases 2 and 1, syphilis was undoubtedly acquired, but in Case 3 the parents are definitely nonsyphilitic and the patient has never been exposed except it be through extragenital routes. There is definite evidence of endocrine disturbances in the families of Cases 3 and 5. Cases 3 and 5 give evidence of hydrocephalus and organic disease of the nervous system, while Case 1 shows a functional condition. All patients are of low mentality except the one in Case 4. Of the patients demonstrating hypofunction, bed-wetting is present in three; segmental distribution of fat is present in all, a low hairline on the forehead, an adrenal "white line," eosinophilia and lymphocytosis, disproportionate torso-leg ratio, changes in the sella turcica, crowding of the teeth on the lower jaw, markedly increased sugar tolerance, low kidney elimination and disturbed metabolism, are likewise present in all. In minor respects the cases, of course, differ somewhat, but these changes perhaps are dependent more on the disturbances of the interrelated endocrine organs and the underlying physical conditions. Particular attention has been given to the pituitary disturbances alone. The investigation of the other internal secretory organs has been left for further study; they have been mentioned here only in so far as they have a direct bearing on the interpretation of the findings.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Sixth Regular Meeting

FOSTER KENNEDY, M.D., *President, in the Chair*

PRESENTATION OF CASE OF INFANTILE NUCLEAR APLASIA OR INFANTILE MOTOR DEFECTS IN THE CRANIAL NERVES. DR. I. ABRAHAMSON.

Dr. Abrahamson considered this condition a congenital and hereditary one, often affecting many generations. Alcohol or syphilis play a small part. This aplasia is often combined with other congenital defects, such as hernias, genital dystrophies, muscle defects and aplasias of other organs. Facial defects are often combined with malformation of the ear or temporal bone. Facial hemiatrophy has been observed, also skin changes. Moebius first described the condition in 1892 as infantile nuclear aplasia. It has been shown, however, by Schultz, Bernhardt, Zappert and others that this is not invariably true. A nuclear lesion need not be primary, even if it can be proved to exist. In peripheral facial palsy all three branches are affected; in supranuclear (central) and in many bulbar palsies the lower two thirds are mainly affected; in congenital motor defects, the upper third is motionless—the lower two thirds still may functionate.

The patient presented, a girl, 4 years and 9 months old, had the characteristic facial expression. She had had nursing difficulties due to the facial involvement and epileptic seizures, another common feature of the condition. The birth had been normal by new gas treatment (not twilight sleep). The baby cried at once. No otitis was reported. At 3 months of age the facial palsy was first noticed. Teething occurred at 7 months, standing at 18 months, walking at 22 months, speaking at 10 months. She is normal in every way except for the facial ocular and lingual palsy. During rest the mouth is not crooked at first, then draws up to the right side. The left aperture is greater than the right. There is flattening of the face on intention; the left facial middle section reacts more quickly than the right. On closing the eyes the face is drawn up on the right side; it is flattened on the left. On laughing, the difference is greatest. There is weakness of both external recti, especially the left. Lateral movements are poor, up and down movements normal. The jaw reflex is more lively on the left. The forehead cannot be wrinkled. There is atrophy of the left side of the tongue with deviation to the left. Faradic irritability is present, and the tip and abductors give normal reactions. The case belongs to the congenital nuclear amyotrophies, and the question raised in Dr. Abrahamson's mind was whether an anastomosis with the spinal accessory nerve would be worth while. He was not optimistic about the operation in this case.

DISCUSSION

DR. FOSTER KENNEDY said that he had seen some similar cases, and thought that it would be unwise to embark on operative procedure. What is obviously present in some of the nuclei is probably present in others, and the grafting operation might be without avail since the graft material might also be affected. The deformity, he felt, would probably become less as time went on.

DR. WALTER TIMME asked whether there had been any diplopia.

DR. ABRAHAMSON answered in the negative. He said that at the time of the patient's birth an epidemic of poliomyelitis had been raging which persisted for some time. The patient's mother had made every effort to keep the baby away from other children on this account. Dr. Abrahamson touched on the possibility of intra-uterine infection, as well as on the possibility of infection in early infancy, but felt that these were practically negligible.

THE WIDENING FIELD OF NEUROLOGY. Address of the retiring president, DR. WALTER TIMME.

Dr. Timme first recalled certain noteworthy meetings of the past two years. He paid special tribute to the memory of Dr. Southard. The radium treatment of nerve tissue tumors, as described by Dr. Ewing and his associates, he considered a contribution of inestimable value. Some lack of advance in purely formal neurology there may have been, due to pessimism concerning the efficacy of treatment of central nervous disease. The age has shown our tendency toward mathematical application to the diagnosis of human ills rather than to their correction. The philosophy of nervous affections, studies of personality, psychoanalytic aspects of nervous disease and analyses of character defect have all had their reflections at the meetings. The recognition of a new disease entity, lethargic encephalitis, however, has urged attention once more to intensive study of the physiology and pathologic anatomy of the central nervous system. In this field the work of J. Ramsey Hunt on the basal

ganglions and the expounding of a new theory of kinetic control, and the monumental work of Tilney and Riley—the first of its kind—on the forms and functions of the central nervous systems, are great achievements.

Perhaps the most important of the activities of the Society during the past years has been the entrance of neurologists into the social-industrial-economic field. The threatened socialism of medicine has brought the physician into direct contact with politics and government. It is of the utmost importance that if the physician is to be controlled by certain laws he should assist in framing them. The public has the right to know why the physician considers himself the guardian of public health rather than various naturopaths, osteopaths, chiropractors and so-called scientist healers. We must meet our wards in their everyday life, guarding them from dangers of employment, of transmitted disease, and diseases of social environmental origin. The Society has established two committees: one on occupational neurologic disease and the other on neurologic standards, or the minimal standards of neurologic training. It is to be hoped that there will be added a legislative committee to make the work of the two other committees effective.

Finally, the Society has not only inaugurated the Association for Research in Nervous and Mental Disease, but this Association has held the first meeting and has presented the findings at a symposium (in this instance on lethargic encephalitis) before a representative gathering. Neurologists of the entire country, and some in Canada and England, have been interested by the association's novel method of procedure; namely, the commission method of jury investigation into the merits of each scientific presentation, in which the proponents are questioned as to their methods of work, their observations and conclusions. This method, which has been used with success, is being made the model of many old time medical societies as the best and most economical in time expenditure, and the most meritorious that has yet been devised in the scientific results obtained.

ADDRESS OF THE INCOMING PRESIDENT. DR. FOSTER KENNEDY.

Dr. Kennedy, taking as his point of departure the findings of the Association for Research in Nervous and Mental Diseases on lethargic encephalitis, proposed to examine available knowledge concerning the routes by which bacteria and toxins gained admission to the central nervous system. The majority of persons with encephalitis had evidence of injury to the mesencephalon, to the striate bodies and to the red nucleus. The first sign of infection other than general malaise was usually diplopia due to disorder of the oculomotor nuclei, a district not considered easily accessible to exogenous poisons. The explanation for the attack and the route followed was not clear: it was merely assumed that the poison or group of poisons responsible for the disease had unknown properties of specific chemotropism for the structures initially damaged. This explanation is not sufficient. When the anatomic and physiologic properties of nervous tissues were first under investigation little or nothing was known of infection, and the constant warfare between the cells and humors of the body and the host of microscopic and ultramicroscopic organisms not even conceived of. The morbid courses of infections are for the first time becoming explicable to us by the portal of the endocrines. A consideration of the pathogenesis of the infections of the nervous system may reveal a unity of morbid process in many ailments clinically unlike, a pathologic synthesis as valuable as the analyses of symptoms customarily made.

The reaction of each person varies with the different physical personalities, possibly in accordance with the different kinds of endocrine balance in each of us. But to detect the minute differences between similar appearing objects requires training. Individual reactions to toxins from without will then become visible—reactions which we now look at but do not see. Further, just as one man differs from another in his reaction to infection, so in a single organism there are a host of unknown and little thought of circumstances which determine the incidence and distribution of the lesions—a patient with Addison's disease has tuberculosis of the suprarenal gland, but one would like to know why tabes mesenterica or fibroid phthisis is not present instead. A statement of such an example shows our wish to be dissatisfied with the mere nomenclature of disease and our desire for basic study of the conditions of liability and resistance to infections in the various tissues and organs of the body.

The most common route of infection of the central nervous system is by the airways, guarded by the cilia of the mucosa and perhaps the mucoid material with which the mucosa is lined. It is probable that ferment action from dead or living bacteria effects the first rent in the epithelium giving access therefrom to the lymphatic and blood systems. Over the respective rôle of these two systems in conveying noxious material there has been much controversy. It would appear that the defensive mechanism of the choroid gland in excluding all hematogenous material unsuited to its purpose, coupled with the anatomic continuity of the lymph system with the cerebrospinal pond, make it more than a working hypothesis that by the perineural and endoneural lymph channels of the cranial and spinal nerves toxins can reach with unfortunate ease the cerebrospinal axis. Attempts at producing brain and cord lesions by intravenous injection of bacteria have failed on account of elective filtration of the choroid plexus whereby large colloid molecules are forbidden access to the central nervous tissue, and the whole group of albuminoid toxins are thus cut off from direct invasion of the brain space. These large molecules can pass easily through the walls of capillary vessels thus breaking from the blood to the lymphatic chain. This permeability of the capillary wall is an important factor in the mechanism of many infections, notably of tetanus. Trismus, one of the earliest signs of the onset of generalized tetanus even when the initial lesion has been in one of the lower extremities, appears to be an evidence of a blood borne toxemia, though the mechanism by which the motor root of the fifth nerve is thus early irritated is not clear. In diphtheritic nerve intoxication oculomotor palsy is a constant feature, and the infection of the third nerve nuclei in epidemic encephalitis is common. This especial susceptibility to react to general infection may not be due simply to delicacy of structure, but may depend on anatomic avenues for invasion as yet not comprehended. The diphtheritic infections studied by Walsh in the Palestine campaign, often taking the form of so-called desert sores, gave nearly pure cultures of Klebs-Loeffler bacilli, and numerous cases of peripheral neuritis ensued. Most important was Walsh's observation that invariably there was an initial local paresis related anatomically to the site of the infective focus, a circumstance suggesting the perineural lymph stream as carrier to the nervous elements. The poliomyelitis virulence of the mesenteric lymph glands of subjects whose blood is innocuous is another circumstance of moment.

Dr. Kennedy described two cases of diphtheritic infection followed by local polyneuritis and correlated with these certain other cases described as acute infective neuronitis. These occurred as a minor epidemic among soldiers in the field and were characterized by fever, peripheral neuritis and signs of

ascending involvement of the spinal roots and ganglions. A constant feature was peripheral paralysis of the face and of the lower muscles of deglutition. The pathologic picture resembled those obtained experimentally by Orr and Rows in their work on lymphogenous infections.

Dr. Kennedy felt that it was a particular detriment to science that medical talent had developed into a variety of fields distinct and separate from each other, and that no adequate headquarters had been evolved where reports could be received and correlated. The modern student received instruction in animal physiology from physiologists who never enter a ward, in chemistry from chemists often without interest in either physics or biology. This segregation of different branches of learning is continued into adult professional life. As a society for the advancement of learning it would be to our advantage to arouse interest in our problems among physiologists and biologic chemists, among pathologists and anatomists, even to include them in our body. Their science would be made humane, our medicine more scientific.

NANISM OF PITUITARY ORIGIN AND OF TALTAUF TYPE COMBINED WITH A POSTINFECTIOUS STRIATE SYNDROME.
DR. WALTER M. KRAUS.

J. G., 28 years of age, was normally born and is said to have been normal up to the age of 18 months. At this time whooping cough confined him to bed for three months. Contractures, involuntary movements and so-called paralysis followed. He had had pneumonia two years ago.

His mother died at the age of 52, of a second stroke. His father died at the age of 52 of cirrhosis of the liver and chronic alcoholism. There were two brothers and one sister, living and well. One brother died in infancy. He is said to have been normal.

This patient presented two pictures, described separately for the sake of clearness. The first was that of dwarfing, nanism.

In Taltauf's case, described in 1891, the patient had been for twenty-one years a military servant, and had subsequently worked as a gardner. He had twice suffered from rheumatic affections of the knees and subsequently on two occasions, from general edema. Three weeks before coming under notice, the general edema returned. He died twelve days later. He showed the following peculiarities: height, 45 inches; horizontal circumference of head, 21 inches. There was slight scoliosis in the upper dorsal region, and marked lordosis in the lumbar region. The external genitals were those of childhood; the left testicle was in the scrotum, the right in the inguinal canal. Necropsy showed lymphatic, glandular and pulmonary tuberculosis, hypertrophy with dilatation of the right side of the heart, fatty degeneration of the myocardium and recent hemorrhage into the pons. The head was relatively big, the face short and broad, with prominent malar bones, the bridge of the nose was broad and saddle shaped; the nose itself was blunt. The neck was short, the thorax convex. The abdomen was hemispherically arched forward. Apart from general edema and other changes stated, there were no noteworthy abnormalities in any part except the skeleton. The thyroid gland was, however, very small and pale red. It is enough to say that as a whole, compared with the normal, the skeleton corresponded to that of a boy of 7 years of age.

Examination of the patient presented at the meeting revealed: Height, 50½ inches, which is the normal height for a boy between 10 and 11 years; lordosis and scoliosis; the genitals of a child. There was almost complete phimosis.

The left testicle was in the scrotal sack, the right in the inguinal canal. The skin was wrinkled and pigmented like that of an old man. Mentality was normal. Joe was graduated from the public schools, read books such as are understood by the normal adult and had a good sense of humor. His voice was high-pitched.

These findings establish the type of dwarfing. His appearance and mentality rule out cretinism. His mentality differentiates him from the Lorain type, who are childish in their mental development. The sexual development rules out the simple ateliosis of Hastings and Gifford.

Dr. Kraus reported a case of a dwarf of this variety about five years ago in which an investigation of the pituitary was made at necropsy. The striking thing was a replacement of nearly the entire gland by brownish material. Practically no normal tissue remained. The patient also had the general edema which Paltauf described. In the case under consideration there is clinical evidence of the pituitary origin of the condition. The patient looked like a miniature adiposis-genitalis of Fröhlich. His breasts were large and soft, his abdomen was pendulous, fat and square in shape at its lower part. There was a large mass of fat above the pubis which looks like the prominent pad seen in the Fröhlich cases. There was no hair on the body except that of the head, eyebrows and lashes. The teeth, the second set, were short and stubby, not spaced and not extensively decayed. The hands and feet were quite small.

It seems apparent, therefore, that there is now good evidence to show that the Paltauf type of dwarf is due to diminution of the function of the pituitary gland and is usually due to a cystic formation.

The second element in the case can be quickly described.

About three months after the beginning of an attack of whooping cough, which occurred when the patient was 18 months old, he had had several convulsions and then lapsed into the state of contracture and choreiform movements of the face and extremities which he now has. The condition was therefore not progressive. He was spastic throughout, more so in the upper extremities than the lower, and more in the right arm than in any of the other extremities. However, he was able to walk. He sometimes had difficulty in starting to speak, sputtering a good deal. However, when undisturbed by involuntary movements, he could speak normally. There was no history of dysphagia, and none had been noticed in the ward. He had frequent seizures in which the arms moved at a slow rate and in a choreo-athetoid fashion, and the facial muscles gave the impression of either laughing or crying. The pupils reacted to light and accommodation. There was almost continual hippus. The reflexes in the upper extremities were normal. The abdominal reflexes were not obtained, possibly due to the flabby abdomen. The tendon reflexes of the lower extremities were normal. The great toe was held in the position of extension as frequently as not and had a greater tendency to do so when the patient was on his back than when sitting. Stimulation of the sole sometimes produced extension of the great toe, sometimes flexion. The Oppenheim and Gordon reflexes were also extremely variable. The thyroid was palpable and hard. There was impaired percussion note over the left lower lobe of the lungs and a few dry râles.

Due to the continual movements roentgenograms could not be taken. The clinical picture was that of a striate level lesion, due to encephalitis following whooping cough.

DISCUSSION

DR. WALTER TIMME said that the cases of Fröhlich's dystrophy that we see are invariably accompanied by endocrinopathic familial disturbances. This case, though it presented some characteristics of Fröhlich history, has no familial history. He was inclined to believe that the whooping cough and synchronous disturbances in the pituitary gland occurring at such an early age would give the rôle of causative factor to the infection.

DR. B. ONUF was not convinced of the superior mentality of the patient. He felt that he was an infantile endocrine type. The expression and attitude indicated it.

DR. KENNEDY thought that the patient might appear infantile emotionally, yet be intellectually mature. His choice of reading matter and skill in the game of checkers would seem to indicate adult mental power.

DR. ONUF referred particularly to the manner in which he followed the proceedings at the meeting, which he considered infantile.

DR. L. PIERCE CLARK agreed with Dr. Onuf in regard to the impression of infantility given.

DR. KRAUS said that the presence of undescended testicles suggested a congenital origin and hence a separate origin for the pituitary disorder, the infection having come on later and having caused the striate level syndrome.

A CASE OF BRAIN TUMOR—CLINICAL AND PATHOLOGIC NOTES.

DR. E. D. FRIEDMAN.

Dr. Friedman reported the case of a 49-year old sea captain who complained of pain in the left side of the face and progressive loss of hearing. The left eye at first became "smaller." Examination revealed that the left pupil was smaller than the right, the left palpebral fissure narrower. There was hyperalgesia in the distribution of the left fifth nerve. Hearing was impaired on the left, and a tumor mass was felt in the neck in the angle of the left jaw. The rest of the neurologic examination was negative. A positive Wassermann reaction indicated antisiphilic treatment. A month later the patient complained of diplopia, and there was weakness of the left sixth nerve. Shortly after this he complained also of persistent objectionable taste resembling that of Jamaica rum. He then had three seizures, each preceded by a sharp pain in the left side of the face, things grew dark before him and he had to struggle to keep from falling. Objects seemed to move to the left; there was no loss of consciousness, but drooling from the mouth and the taste persisted. The attacks, which came about a week apart, lasted from five to ten minutes. Four months after the first admission the patient felt that his symptoms were considerably worse. He had lost vision in the left eye; the eye was very prominent, and the swelling of the neck was larger. There was complete paralysis of the third nerve on the left, except that the pupil was small. Fibrillary twitchings were noted in the motor fifth nerve. The sensory fifth nerve was completely paralyzed on the left. There was no herpes; the sixth nerve was paretic. The seventh was normal on voluntary innervation, although there was a slight drooping of the left angle of the mouth. There was no nerve deafness on the left but the tuning fork was lateralized to the left. There was percussion tenderness in the left frontoparietal region. No brain stem phenomena were observed. The patient became rapidly worse and died after a period of neuromuscular conjunctivitis and evidence of retrobulbar pressure, rather than choked disk, on the left.

Roentgen-ray examination of the chest showed no metastatic foci in the lungs or chest wall. Roentgen-ray examination of the head was negative. The general physical examination was negative, with the exception of considerable edema of the palate. Blood pressure was normal. The Wassermann test of the blood was positive. The spinal fluid was clear under fair pressure, 254 cells, globulin was positive, the colloidal gold curve normal, and the Wassermann reaction was positive. The visual fields on the right were normal. There was no aphasia.

Because of the positive serologic findings and the involvement of the nerves at the base of the skull, the patient was subjected to intense antisyphilitic treatment. In spite of this, he rapidly lost ground. The diagnosis was then changed to tumor involving the middle fossa of the skull on the left. The behavior of the left pupil was explained on the basis of an injury to the dilator fibers of the third nerve. The slight drooping of the angle of the mouth on the left was thought to be due to the sensory loss in the face with resulting diminution of muscle tonus. The seizures were looked on as probably gustatory fits which found their explanation in the adhesions between the tumor mass and the tip of the temporal lobe. Posterior fossa neoplasm was excluded by the absence of nystagmus, nerve deafness, cerebellar signs and true papilledema.

Necropsy revealed a tumor roughly occupying the middle two thirds of the middle fossa on the left, overgrowing the pituitary body and overlapping part of the lesser wing and the basilar portion of the sphenoid and some of the petrous portion of the temporal bone. It had surrounded the third, fourth, fifth, and sixth cranial nerves at their points of exit from the skull, and a protrusion forward into the orbit had surrounded the left optic nerve. A further projection of the tumor extended through the foramen lacerum into the pterygoid fossa for about 2 cm., where it ended abruptly, no connection being made out between it and a nodular mass on the left side of the neck about 8 cm. in length. Both middle ears were full of turbid fluid and the left maxillary antrum contained pus.

Histologically the growth did not conform to sarcoma or endothelioma of the dura, these being the types of neoplasm most frequently found in the middle fossa, and which not infrequently metastasize in the neck. The microscopic preparations of the nodular mass in the neck, however, showed it to be an endothelioma of the variety occurring primarily in the lymph glands, and as no further tumor was found on careful examination of the rest of the cadaver, the somewhat unusual conclusion must be reached that the intradural growth was secondary to that in the neck. The histologic findings bore out the clinical deductions to an unusual extent. The deafness of the left ear was doubtless occasioned by sepsis and not nerve defect. The exophthalmos may have been due to pressure of the tumor forward as it did not invade the orbit. Although the brain substance did not appear to have been invaded, pressure on the temporal lobe may have been sufficient to account for the gustatory sensations.

EFFECTS OF PROHIBITION AT BELLEVUE HOSPITAL. DR. JOHN W. BRANNAN.

Dr. Brannan (by invitation) reported that with the going into effect of the national prohibition amendment the number of admissions to the alcoholic wards was markedly lessened. When the Volstead law went into effect the same decrease was expected, and for the first two months in 1920 there was a

marked decrease, but shortly thereafter the admissions began to increase, and the latter half of 1920 gave a total of 1,386 admissions, or only eighteen less than the total admissions for the first half of 1919. The totals for the years, however, showed 2,211 for 1919 and 2,312 for 1920. It had been observed that the admissions had been gradually decreasing since the entry of the United States into the war, and a chart drawn up to cover the admissions since 1917 gave the marked decrease in totals from 5,714 in 1917 to 2,439 in 1918. America's entry into the war in April, 1917, was followed by a drop from 628 to 547 in May, 413 in June, 345, 376, 405, 358, 295 and 372 for the succeeding six months.

An improvement in general conditions is evident from the fact that the number of patients showing symptoms of chronic poisoning (twenty years ago one of the most important factors in the history and condition of patients in all wards of the hospital) has strikingly decreased.

In order to explain the increase in admissions in the past six or eight months, Dr. Brannan thought that the unconcealed operation of the innumerable saloons and the apparent winking of authorities in charge of enforcing the government laws might be adduced as sufficient cause. In handling the alcoholic patient in years past a repeater would occasionally be sent to court, where the magistrate would usually rule that drunkenness was not a crime and would discharge the offender. Recently it was suggested that an alienist should testify to the patient's condition and accompany him to court to try to have the frequent offender committed to the workhouse. Dr. Brannan saw Mr. Justice MacAdoo to determine whether the patient could be made to testify under the Volstead act as to where the liquor was obtained, and was told that the courts had no authority to compel the giving of such information. Dr. Brannan next went to Col. Cathey, United States attorney, with the suggestion that the offenders be sent to the Island on charges of vagrancy. In this case he was told that any magistrate would send the patient home to support his family. The records of the patients admitted to the hospital showed that bachelors were usually in preponderance. Finally Dr. Brannan went to the police department, and the inspector intimated that he might be able to do something with the saloons that were operating openly, a list of which Dr. Brannan supplied him. No report had come in of any action at the time of the meeting. Dr. Brannan said that he had come to the conclusion that federal aid would not be forthcoming, and that local authorities would have to act. In fact, the United States attorney said to Dr. Brannan that enforcement was only a small part of the work to be done by this department.

REMARKS ON THE DIMINISHED THERAPEUTIC USE OF ALCOHOLIC STIMULANTS AT BELLEVUE HOSPITAL. BEGINNING BEFORE PROHIBITION. DR. JOHN W. BRANNAN.

Dr. Brannan presented charts to show the decrease in prescribed whisky in Bellevue Hospital from an average of 0.59 ounces a day per patient in 1903 to 0.06 ounces in 1920. This reduction has been observed in other hospitals and has been progressing slowly, almost without attracting attention. It used to be customary to give alcoholic stimulation fairly generally in the general surgical wards, and even to the alcoholic patients. The latter now receive no whisky ration, and this may possibly account for their exceedingly short sojourns in the hospital at the present time. But even the general medical and surgical wards show a marked decrease, although this decrease varies somewhat according to the superintendence. A marked increase in the per

capita consumption in January, 1919, the time of the dying out of the influenza epidemic, is undoubtedly to be explained on the ground of the vast number of influenza patients in the hospital at that time. Here again those in charge of wards showed a variability in the doses given, ranging from only a fraction of an ounce to 16 to 24 ounces in some wards during the height of the epidemic, October and November, 1918. This change has been going on undirected by any outside or inside force at the hospital, and has apparently escaped general attention.

DISCUSSION

DR. GEORGE O'HANLON (by invitation), in opening the discussion, said that he could not add much encouragement to the hopefulness of sending bachelors to court in an effort to have the alcoholic offenders committed, since they all proved to have dependents. At a hospital conference the question of the use of alcoholic liquor in the hospital was brought up. Practically all the representatives at the conference had had the same experience. One hospital was noted to have been prescribing extensively since the beginning of prohibition. No reason for this could be found except the attitude of the staff. The Philadelphia Hospital had had the same experience as Bellevue. They had been able to close the alcoholic wards. During the last three or four months an increase in patients has led them to consider reopening the wards. The Cook County Hospital in Chicago reported the same experience. In short, with the coming of prohibition there had been a large diminution in the number of persons applying for admission to the hospitals, as well as to the Municipal Lodging House. The employment problem had probably had an effect here also. An increase had been noted in the last few weeks; four years ago the admissions were 2 300, recently the number went down to 35, and is up to 350 at present. Commissioner Coler reported also a decided decrease in applications to the child welfare bureaus since the advent of prohibition. No recent increase had been noted in this branch of public welfare work. During the first few months of prohibition there was a decrease in the ambulance calls, although the cause for this cannot be determined. There is a marked decrease from the number of calls received three or four years ago.

DR. C. B. CRAIG asked whether the beds vacated during the period when the admissions were few were filled with legal types of illness. Dr. Brannan said that they were.

DR. O'HANLON said that the Bellevue authorities were glad to be able to close the wards formerly used for alcoholics and thus release part of the nursing staff. The wards formerly assigned to alcoholic patients are now used for mental defectives.

DR. G. H. KIRBY said that though Dr. Brannan's figures showed some recrudescence in alcoholism during the last few months, this may be regarded as insignificant when one compares the number of cases now appearing at Bellevue to the number a few years ago. He thought there was no doubt that the habits of people have changed in recent years and that the prohibition amendment was the culmination of what has been steadily advancing for a decade or more. The emotional reaction of the population to the war as expressed in terms of alcoholism was interesting. There had been a steady decline in the number of cases of alcoholism and alcoholic psychoses at Bellevue Hospital for some years until just before the United States entered the war, when there was a perceptible rise. Following the declaration of war

by the country a marked drop occurred which, so far as alcoholic insanity was concerned, has continued without further interruption.

DR. J. A. HARTWELL (by invitation) said that alcohol has been proved to be a depressant rather than a stimulant, and all surgeons were becoming more and more convinced that to use it was actually harmful. The time was approaching when it would not be used either by rich or poor.

DR. KENNEDY said that the general conclusion has been that the feeble-minded were more alcoholic than normal people; but Dr. Pearce Bailey's observations showed that the more feeble-minded people were, the less they drank.

Book Review

THE FORM AND FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM. An Introduction to the Study of Nervous Diseases. By FREDERICK TILNEY, M.D., PH.D., Professor of Neurology, Columbia University; Attending Neurologist, the Presbyterian Hospital, and the New York Neurological Institute; Consulting Neurologist, Roosevelt Hospital, New York, and HENRY ALSOP RILEY, A.M., M.D., Associate in Neurology, Columbia University; Associate Attending Neurologist, New York Neurological Institute; Attending Physician, Neurological Department, Vanderbilt Clinic, New York; with a foreword by GEORGE S. HUNTINGTON, Sc.D., M.D., Professor of Anatomy, Columbia University. 591 figures containing 763 illustrations, of which 56 are colored. Price, \$12.00. Pp. 1020. New York: Paul B. Hoeber, 1921.

"The function of the teacher is to teach, and to propagate the best that is known and taught in the world." (Matthew Arnold.)

In the words of the authors, "This work is designed to fill the gap between morphology and the practical requirements of clinical medicine. It aims to visualize the living nervous system, to make accessible an appreciation of its vital relations to the functions which go to make up life, as well as the defects in these relations which result in disease." This is the object of a work which is called, "An Introduction to the Study of Nervous Diseases." It is, however, a profound exposition of the anatomy and physiology of the central nervous system, and an impressive demonstration of the fact that a philosophic understanding of the subject is a living part of clinical neurology.

The book is divided into fifty chapters and is profusely illustrated. Chapter 1 deals with the importance and evolutionary significance of the central nervous system and with the two major mechanisms which control the organism: the somatic, which keeps the animal in contact with its environment, and the splanchnic, which controls its vital processes.

The account of the embryologic development of the central nervous system is unusually clear and easily understandable, and is illustrated by drawings which are mostly original reproductions of reconstructions by the authors.

The nerve cell—the unit of structure of the nervous system—is described in great detail in chapter 4, and the neuron theory is explained in chapter 5. The authors follow Cajal in their account of the different varieties of nerve cells. Like Cajal and Bechterew, they point out that the typical nerve cell of most vertebrates has no centrosome, which explains the absence of reproduction and repair in the cells of the central nervous system. In the neuroglia cells, however, a centrosome can regularly be observed, and these cells have a high degree of reproductive power. The authors' account of the development of a centralized nervous system which culminated with the appearance of the psycho-associational neurons in the final and highest central nervous mechanism of man, is an unusually lucid description of the different stages which led up to the highly developed organization of the human species. The story is a most interesting one: the most primitive form of the nervous system is diffuse, and local in its action, and the development from this to the most highly specialized nervous mechanism has occurred as the result of elaboration and development of the properties of sensitiveness, conductivity and correlation.

The special anatomy of the different parts of the central nervous system is begun with the sixth chapter. Chapters 6 to 8 deal with the methods by

which the human spinal cord is exposed for study, with the relations of the cord to the surrounding structures, its general anatomy, coverings and blood supply.

The histology of the cord segment, the arrangement of cell groups in the gray matter and the differences between the gray and the white matter, both as regards structure and functions, are then described. The grouping of cells in the gray matter is well represented by a large series of diagrams. We know of no better description and no more striking series of illustrations of the final common pathways of impulses through the spinal cord than those given in this book. The student will have no difficulty in comprehending how the common pathway has been gradually evolved—from the simplest control of a muscle by a nerve cell up to the complicated mechanism of voluntary and inhibitory control.

Finally, the main syndromes of the gray and white matter of the spinal cord are described and are illustrated by histories of actual cases. These histories are to the point; the clinical findings are fully presented with the interpretation of the symptoms and physical signs and an anatomic analysis in each instance. The study of these clinical records and their analyses is not only an exercise in anatomy and physiology, but is a striking demonstration of the fact that a thorough knowledge of the form and functions of the central nervous system is the foundation of good clinical neurology.

Six chapters of the book are devoted to the medulla oblongata, and we think that it is no exaggeration to state that the treatment of the subject matter of these chapters is unique. Tilney and Riley give an account of the reasons for the complexity of structure and functions of this part of the nervous system which must impress every reader with the authors' philosophic understanding of the factors of comparative anatomy. The different chapters deal with encephalization and a general view of the medulla; relations, surface appearance and anatomy; internal structure and histology; functional significance, and syndromes of the medulla. As in the chapters on the spinal cord, and as in other parts of the volume, instructive clinical histories with clinical and anatomic analyses are given.

The chapters on the cerebellum are among the best of this unusual book. The general view of the evolutionary significance of this suprasegmental portion of the central nervous system is well given and especial attention is directed to the phyletic constancy of the organ which, as we now know, dominates the proprioceptive functions of the body. The authors give a brief review of the appearance of the cerebellum in the several classes of vertebrates in order to emphasize the rule that the wider the range of motor activity possessed by an animal, the more highly developed is the cerebellum. Then follows a description of the functions of and localization in the cerebellum in which the history of the development of medical knowledge is traced from the earliest researches of Vulpian and Magendie to the recent work of André-Thomas, Bolk, Elliott Smith, Van Rijnberk, and the clinical studies of Luciani, Lewandowsky, Bárány, Weisenburg, Gordon Holmes and many others. The authors believe that synergia is the principal function of the cerebellum and that synergia is dependent on two factors—"the establishment and maintenance of synergic muscular units throughout the body, and the establishment and maintenance of coordination between the synergic units in the performance of complex acts." In other words, as Herrick puts it, the cerebellum controls the more perfect coordination and integration of the somatic motor reactions and strengthens these reactions. While this point of view is not yet generally

accepted, it is—as the authors claim—a common ground on which all may stand and on which the many differences in point of view may be finally conciliated.

More than 300 pages of the book are devoted to the end brain. This portion of the work is very complete and is a clear exposition of the points of view not only of the authors themselves, but also of most other writers. Separate chapters deal with the development and comparative morphology of the cerebral hemispheres, with craniocerebral topography and cerebral measurements, with the coverings of the brain and the craniocerebral circulation.

Then follows a detailed account of the cerebral cortex and its cell layers and of the medullary substance, the functional significance of these parts, and the principal syndromes due to lesions of the cortex or the central gray matter.

“As the end brain gradually increased in size, new capacities for experience were added to the nervous system, and the advances made themselves felt in animal behavior—experience being the sum total of the sensory impressions received by the end organs and correlated in the brain, and behavior being the expression of this total. The cerebral cortex was once entirely devoted to the correlation of olfactory and gustatory impulses but finally it became the area wherein the most complex correlations and associations of the various types of sensory perception occurred, thereby constructing the foundations for the higher psychic faculties and the individualistic behavior of man.” While the cortex thus controlled individualism, one of the great central ganglions—the corpus striatum—still exercised a control over generic behavior. The reviewer has quoted this portion of the text because it is an admirable summary—in one short paragraph—of the underlying basis of human behavior and human conduct located in the human cortex and the central ganglions.

The description which the authors give of the internal nuclei of the cerebral hemispheres, and especially of the corpus striatum, is a classical one. The corpus striatum in mammals is to be regarded as the homologue of the basal forebrain ganglions in the lower vertebrates, and is a motor pathway to the higher parts of the central nervous system. With the development of the cortex, a new motor center and a new motor pathway was developed so that the somatic muscles came under a dual control—one paleokinetic, dependent on the primitive connection with the corpus striatum, the other neokinetic, connected with the cortex.

Following the views of Ramsay Hunt, the authors make a distinction between the paleostriatum (globus pallidus) and the neostriatum (putamen and caudate nucleus); they believe that the neostriatum made its appearance at an evolutionary epoch when new environment required new and special motor adaptations. The corpus striatum belongs, therefore, to the motor system; its function is to regulate automatic associated movements and to control the tone of the striated muscles of the body.

The putamen contains some elements of the paleostriatal system, and when it is diseased, the symptoms are similar to those of paralysis agitans except that muscle hypertonus is marked. The caudate nucleus belongs to the neostriatum and inhibits the activity of the globus pallidus.

If the globus pallidus is especially affected by disease the symptoms are those described by Ramsay Hunt as the paleostriatal syndrome—increase of muscle tone, suppression of normal automatic movements, tremor, without pyramidal and somatic sensory and splanchnic disturbances.

Progressive lenticular degeneration, especially if it affects the putamen, gives rise to the well-known syndrome of Wilson's disease, while lesions of both putamen and caudate nucleus cause symptoms among which double athetosis is prominent.

While the authors thus relegate definite functions to different parts of the corpus striatum, they acknowledge that our knowledge is still limited and that much will be changed and added. Without entering on a discussion of this subject, it seems to the reviewer that, entrancing as are these points of view of the functional significance of the corpus striatum, our knowledge is not yet based on a sufficient number of facts to permit acceptance of definite functions for the different parts of the corpus striatum.

In the chapters on the cortex and cerebral localization, a complete description of the cell groups in the various parts of the cortex is given. The authors divide the cortex into the following areas: the precentral or motor area, whose functional significance is volitional motor control and inhibition; the intermediate precentral or psychomotor area, in which the motion formulae for skilled acts are constructed and retained and lesions which (on the left side in right-handed persons) give rise to motor apraxia and its special forms of motor aphasia and agraphia; the postcentral or somesthetosensory area, which is the primary receiving station of all somesthetic impulses which enter into consciousness (thalamocortical pathways of Head); the intermediate postcentral area, which serves for the memory registration and apperception of impulse intensity and relativity and for the synthesis of many sensory impressions which result in the stereognostic, barognostic and other senses; the calcarine or visuosensory, the occipital or visuopsychic, the audiosensory, and the auditopsychic areas; the limbic area and the rhinencephalon; the insular, parietal, frontal and prefrontal areas.

The boundaries of each of these areas is carefully outlined; the cells and fibers are compared with those of other parts of the cortex; the functional significance of each area is—wherever knowledge permits—made clear; and the symptoms which result from disease or injury are given.

This part of the book, dealing as it does with many complex questions of normal and abnormal psychology, is clearly presented and is of absorbing interest, and the explanations given are based on most advanced views, and compare favorably with larger monographs such, for example, as that of von Monakow.

Some of the features of the volume by Tilney and Riley have been mentioned to emphasize that this is a most unusual book. It is more than an anatomy of the central nervous system, for the study of function is everywhere interwoven with that of structure. The text combines anatomy, physiology and semeiology. It is evident that the authors believe—and rightly—that these belong together.

The style of the authors is clear and readable and never involved. The illustrations are excellent. Most of them are original and really illustrate the statements made in the text, although no reference to any figure is made in the text. The diagrams of the course of fiber tracts are worthy of special mention, as are those of serial sections of the brain. In the figures which accompany the clinical histories, the areas of disturbed function are marked in different colors, and in each instance a cross section is added to indicate the location of the lesion.

The book is well set up and printed, and is a volume of which any publisher might well be proud. There are some typographical errors which will

surely be corrected in a succeeding edition, and some of the illustrations of cross sections have lost in detail through reproduction on account of too heavy printing and too much printer's ink.

For the undergraduate medical student, this volume is valuable both for study and for reference. For the more advanced student it contains much information and much food for study and for thought. For the mature neurologist, however, and for every one who desires to acquire a deeper insight into the form and functions of the central nervous system, from the combined points of view of embryology, morphology, comparative anatomy and symptomatology, it is a book which should be consulted carefully and often.

Original in title, unique in the handling of the subject, this volume is certain to occupy a high place in the literature of the nervous system.

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FAMILY SPASTIC PARALYSIS OF SPINAL TYPE ON A HEREDOSYPHILITIC BASIS

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It is only recently that clinical histories have been published concerning patients suffering from heredosyphilis who have developed a syndrome identical to spinal spastic paralysis. Such cases are comparatively rare. Nonne,¹ in the last edition of his book, was able to report only about ten cases, including his own private ones. I have therefore thought it opportune to report the clinical histories of three such cases, occurring in two brothers and a sister, the offspring of a syphilitic father.

REPORT OF CASES

CASE 1.—History.—G. V., aged 54, a tradesman, at the age of 22 suffered from what was diagnosed as soft chancres of the penis. Married soon after, his first-born, a son, remained in good health. At intervals of two years a second son, a daughter and a third son were born. The fifth child, 14 years old, seemed slightly deficient, but was free from the syndrome of the three cases reported. The mother died four months ago from cardiopathic disease.

At the age of 46, the father began to complain of sexual impotence and involuntary urination. Lancing pains in the lower extremities, a sense of oppression in the chest, incomplete emptying of the bladder, crises of dry cough with spasmodic dyspnea had only recently been complained of. He also complained of loss of memory.

Examination.—(November, 1920). Examination revealed: no motor or sensory disturbance of any of the cranial nerves; slight hypotonia of all four extremities; inequality of patellar and tendo Achillis' reflexes, both being weaker on the right side; normal upper extremity reflexes; the corneae were glistening, pupils punctiform, not reacting to light and reacting sluggishly to accommodation. There was hypalgesia along the ulnar surfaces of the forearms. There was no tenderness over the nerve trunks or grooves. Girdle hypalgesia was more marked on the right side, extending from the fourth intercostal space to the umbilicus. The Romberg sign was present. There was appreciable disturbance of locomotion. Specific senses were normal. The lymphatic glands on the right side of the neck were enlarged. The heart had a marked dilatation of the right ventricle with an aortic diastolic souffle; arteriosclerosis was present. The blood Wassermann test was positive. Lumbar puncture was refused by the patient.

1. Nonne: Syphilis und Nervensystem, Ed. 3, Berlin, 1917.

CASE 2.—History.—This patient was the second son of G. V. He had retarded intelligence from infancy, but no definite syndrome developed until he was 16 years old, when he gradually began to complain of a slight amblyopia of the right eye which progressed to almost complete amaurosis within three years. While blindness was coming on in his right eye, amblyopia of the left eye gradually supervened, progressing to blindness in about two years. A concomitant slow and progressive weakness of the lower extremities was noted, with sensations of constriction in the limbs. He was, however, able to walk without assistance.

Examination.—There were no disturbances of the cranial nerves, with the exception of the optic nerve. Active and passive movements of the neck and upper extremities were normal; speech was normal. Muscular nutrition of the lower extremities was normal; passive movements met some resistance; active movements were correctly carried out, though sluggishly. In the act of walking the feet were only slightly raised and the knees slightly flexed (paretic-spastic gait). The tendon reflexes of the lower extremities were all active; there was no Babinski sign. The pupils were equal and sluggish to light. Sensory reactions and specific senses were normal. Right Eye: There was a soft complete cataract of this eye; the fundus could not be explored; light perception was normal at 3 meters. Left Eye: There was an incomplete soft cataract of this eye, also; the fundus showed atrophy of the optic nerve (post-neuritic); several areas of chorioretinitis in the periphery.

CASE 3.—History.—A daughter of G. V., aged 22 (third born), experienced no disturbances until 16 or 17 years of age when she began to notice a sense of weakness and constriction of the lower extremities which slowly progressed until she not only dragged her feet and barely flexed her knees, but was so weak that, after taking a few steps she had to sit down, requiring assistance in order to prevent her from falling. There had never been any vesical or rectal disturbances, but she suffered from severe ozena. She had not received treatment.

Examination.—The cerebral nerves were normal; speech was, however, halting in type (bradyarthria). The upper extremities were normal. The lower extremities showed resistance to passive movements; active movements were all slow and incomplete, especially those of the feet. In walking, the feet were only slightly raised and the knees slightly flexed; she stood mainly on the toes; the radial reflexes were absent; only the right epigastric reflex was present; both the abdominal and the left epigastric reflex were absent. Patellar and tendo Achillis' reflexes were active; the plantar reflex on the right side spread out in fan shape. The Babinski sign was absent. The pupils were of medium size with a tendency to myosis; they reacted slightly to light. There was normal sensibility of the specific senses and sphincters. The blood Wassermann reaction was + + +. Mental deficiency was present; ideation was infantile; there was fairly well marked apathy even as regarded her vegetative functions. She did not evince any interest in her case. Memory was poor; perception was correct though rather limited to elementary demands.

CASE 4.—History.—A son of G. V., aged 20 (fourth born), from childhood showed marked signs of deficiency. At 16 he began to develop weakness of the lower extremities with rigidity, progressive in type.

Examination.—The cranial nerves were normal as were also the upper extremities. There was diminished strength in the lower extremities with abnormal resistance to passive movements. Abdominal, patellar and tendo

Achillis' reflexes were greatly increased. There were no rectovesical disturbances and no appreciable changes of general sensibility or of specific senses. Blood and spinal fluid tests were refused by the patient. Mentally the same defects were found that were apparent in his sister, but more pronounced; retarded perception, poor memory, apathy, stubbornness, and poor reasoning power.

Conclusions.—It is evident that the father was a tabetic patient; the history and clinical findings were typical. With the exception of the first-born, all of his children were more or less deficient; all three, at about the same age (at puberty) began to suffer from progressive motor disturbances of the lower extremities terminating in the syndrome of family spastic paralysis (spinal type). One of the brothers also suffered from optic atrophy, choroiditis and double cataract.

CASES IN THE LITERATURE

The following similar cases are reported in the literature, most of them in Nonne's book:¹

Friedmann² reports the cases of two children, 10 and 15 years old, who had spastic spinal paralysis on a probable syphilitic base. In the first case there was complete recovery after two attacks, under anti-syphilitic treatment; the second patient also had two attacks with complete recovery after the first one and only improvement after the second.

In Hoffmann's³ case the mother had a history of syphilis. The boy developed spastic paralysis at 12 years of age with arrested mental and physical development. He had mydriasis, paresis of accommodation and pupillary rigidity.

Mendel⁴ reported a case of spastic spinal paralysis on a syphilitic basis manifested at the age of 5, with progressive weakness and obtunded sensibility of the lower extremities. Slight ischiuria was also present.

Sachs⁵ reported the case of a patient with a tabetic mother with a history of two abortions and the early death of three infants. The patient developed the spastic syndrome at the age of 5 with retrogressive mentality. In this case there was also found a spastic paresis of the upper extremities with pupillary disturbances one year later.

Luzenberger⁶ found the syndrome in two brothers whose parents probably had suffered from syphilis.

2. Friedmann: Ueber rezidivierende sogenannte spastische Spinalparalyse im Kindersalter, *Deutsch. Ztschr. f. Nervenhe.* **2**: 1892.

3. Hoffmann: *Neurol. Centralbl.*, No. 13, 1894.

4. Mendel: Quoted by Nonne.

5. Sachs: *The Nervous Manifestations*, *Am. Med. Bull.*, 1896.

6. Luzenberger: Quoted by Nonne.

Vizioli⁷ reported the cases of three brothers and a sister; the father was syphilitic. All four had chronic degenerative anomalies and Hutchinson's teeth. Only one could talk clearly; the others had disturbances of articulation. All four had normal births at term and appeared normal until they began to walk, when rigidity with the typical adduction and internal rotation of the lower limbs was noticed; it was also difficult for them to sit down. Spasticity of the upper extremities was less marked and there was also a certain degree of rigidity in the muscles of the face and neck and of deglutition. The reflexes increased variably. All manifested some degree of intellectuality.

Königstein⁸ reported a case in which there was syphilis in both parents. The patient at the age of 9 showed slight mental defects, spastic paresis of all the extremities and rigid pupillary reflexes.

In one case reported by Nonne the mother had a chancre of the lip two years before her marriage. The son, when 10 months old, had a roseola and psoriasis plantaris, but otherwise developed normally until the age of 4, when disturbances of locomotion manifested themselves. Spastic gait, ankle clonus and the Babinski sign were present; pupils were normal; there was a slight grade of cranial rachitis.

Nonne also reports a case in which the father was syphilitic. The patient was sickly at birth. He began to walk at the age of 3 and from then on retrogressed mentally. Examination showed subnormal bodily development, stupid expression, left convergent strabismus, spastic paresis of the lower extremities, ankle clonus and a Babinski sign. There were rigid reflexes of the left eye, weak reflexes of the right eye with bilateral partial optic atrophy and multiple foci of choroiditis.

In a third case reported by Nonne the father was syphilitic. The child was born prematurely (7 months). It was weak and began to talk rather late. First dentition was also retarded. On account of the spastic paresis of the lower extremities, it never walked. The child looked like an imbecile. There was slight left convergent strabismus. The cranium was slightly microcephalic. The teeth were carious. Speech was normal. No sensory and sphincteric disturbances were found.

In a fourth case reported by Nonne the mother had given birth to four older children who had all died in infancy. The patient at 9 years of age had weakness and rigidity of the extremities; he was queer, bad tempered and avoided other children. The gait was

7. Vizioli: Quattro casi di diplegia spastica familiare infantile eredo-sifilitica, *Ann. di Neurol.* **12**: 1898.

8. Königstein: Ein Fall von luetischer Spinalparalyse und reflektorischer Pupillenstarre, *Mitt. d. ges. inn. Med. u. Kinderh.* **9**:246, 1910.

pareticospastic, slight foot clonus and a bilateral Babinski sign were present; the right pupil was rigid, the left reacted only to convergence. The patient suffered from general weakness and was somewhat imbecile. Wassermann reaction: blood +, spinal fluid +, globulin positive. In the mother the Wassermann reaction was pronounced.

Artigalas⁹ reported the cases of three brothers all displaying spastic phenomena and muscular pseudohypertrophy. The syndrome gradually diminished under mercurial treatment.

Finizio¹⁰ reported the cases of two children of syphilitic parents who began to show, at the age of 3, pareticospastic phenomena. One child also exhibited bradylalia, and the other deficiency of the extrinsic eye muscles.

Déjerine¹¹ reported the case of a child who at the age of 8 began to show locomotor disturbances which slowly resulted in contractures. The thighs and legs were extended, while plantar flexion coexisted. Mental development was retarded. The pupils were rigid and unequal. The spinal fluid showed a profuse lymphocytosis.

SUMMARY

The preceding cases allow me to form a clinical grouping of the syndrome observed in each case.

The first signs of the spastic spinal syndrome due to heredosyphilis may manifest themselves just as the child begins to walk; locomotion may begin normally or be retarded; in other cases the first signs appear after the child has learned to walk properly. Thus, Nonne's first case had its onset when the patient was 4 years of age, Mendel's at 5, Déjerine's at 8, Hoffmann's at 12 and mine at puberty.

The motor disturbances—spastic paresis—may be limited to the lower extremities, as in Nonne's second and third cases, in Mendel's case and in my cases; or they may extend to the upper extremities, as in Sachs', Vizioli's, Königstein's and in the first and fourth cases of Nonne. The paresis and spasms were sometimes uniform, rarely severe enough to prevent the patient from standing up (Nonne's second and third cases). The spasms have a gradual onset and sometimes attain a severe degree of rigidity; sometimes even extension contractures of the thighs and legs and plantar flexion of the feet have been present. In one case spasm of the muscles of the neck was found. Reports on the condition of the lower extremity reflexes are

9. Artigalas: Quoted by Sandri in *La sifilide ereditaria del sistema nervoso*, Milan, 1911.

10. Finizio: Quoted by O. Sandri in *La sifilide ereditaria del sistema nervoso*, Milan, 1911.

11. Déjerine: *Paraplégie spasmodique de l'enfance*, Soc. de Neurol. de Paris, 1904.

incomplete; generally speaking, they are exaggerated (my own cases). Foot clonus may be present (Cases 1, 2 and 4 of Nonne). The Babinski sign was seldom present. In many patients the pupils did not react to light (Déjerine, Hoffmann, Sachs, Königstein, and the fourth case of Nonne). Anisocoria is less frequent; in my cases, only the second son had this symptom, but he also had a bilateral cataract and optic atrophy, the latter condition being also a feature (unilaterally) in Nonne's second case. In two cases there were left convergent strabismus and weakness of the external ocular muscles (Finizio). General sensibility is usually well preserved. In one case, rectovesical disturbances were recorded, resembling Erb's syphilitic paralysis. Disturbances of articulation (bradyarthria) were noticed in a few cases.

Psychic disturbances were common, but none seemed to attain a severe degree. My own patients all manifested varying degrees of imbecility—deficient attention, poor ideation, weak reasoning power, apathy, etc. Some patients, notably Vizioli's, had a normal mentality. The Wassermann reaction was positive in my cases and in Nonne's fourth case; it was negative in the spinal fluid of Nonne's second patient. Syphilis was assumed as the result of specific therapy (Artigalas) or after examining the spinal fluid (Déjerine). Syphilis had undoubtedly infected either the mother (Hoffmann, Sachs, first and fourth cases of Nonne) or the father (Vizioli, fourth case of Nonne and my cases) or both parents (Luzenberger, Finizio). This does not mean that in any of these cases one of the parents was immune; it is likely that an important factor in the production of this disease is the lack of antisyphilitic treatment in the parents. In Hoffmann's case, the mother had had two abortions and one child who lived only fourteen days; in Sachs' case there had been two abortions and three children who died in infancy; in Nonne's fourth case the mother had lost four infants. In my cases, the children were born shortly after the father had acquired syphilis, for which he had taken no treatment. Many authors state that there were no degenerative or heredosyphilitic stigmas in their cases; Vizioli alone reports cranial anomalies and Hutchinson's teeth. Often the spinal spastic paralysis assumes the family type (Vizioli, Luzenberger, Finizio and my own cases). It is not unreasonable to suppose that many of the cases of family spastic paralysis, described at a period when syphilis was not carefully considered, are really due to heredosyphilis. In the cases of Tooth and Tambroni,¹² in which syphilis was not even mentioned as an etiologic factor, family spastic paralysis began in infancy or in the early juvenile

12. Tambroni: *Sopra un caso di tabe spastica famigliare*, Riv. sper. di freniat., 1897.

period; at present there are cases of this syndrome based on heredosyphilis (Luzenberger, Vizioli, Finizio and my cases).

— It is hard to say under what conditions heredosyphilis will produce spinal spastic paralysis rather than some other type of cerebral or spinal disturbance. The syndrome, or to be more exact, the various disturbances more or less constantly associated with the dominant symptom of spastic paralysis, suggest the conception of a primary morbid process of the encephalon, particularly of certain cortical areas and of their associated fibers. In some cases it is difficult to separate these types from Little's syndrome, because the pareticospastic motor disturbances only appear after the child has begun to walk (Vizioli, Nonne's second, third and fourth cases). Nevertheless, even in the cases of late development, in which the first symptoms appeared at the ages of 4 or 5, or even at 12 to 14 years of age, it does not seem to me that one should deny a close similarity of the syndrome in question and that of the congenital spastic paralysis of Little. The fundamental pathology of Little's syndrome lies in a deficient development of the pyramidal tracts which, arrested in the cervical segment or in the dorsolumbar segment, produce, respectively, a spastic tetraplegia or a paraplegia. One can extend this conception (the possibility of Little's syndrome being the same as that of family spastic paralysis) and admit that in the late cases of the disease in question the pathogenic element (syphilitic poison) affects the extreme end of the corticospinal protoneurons, pyramidal neurons, which are already developed, in order to understand that this will lead to an early decay—apparently primary—of the distal portion of these neurons. But in order that this may occur, it is necessary to fall back on a postulate, that is, that the neurons arising from the cerebral cortex, and especially those forming the corticospinal bundles, are so delicate that slight disturbances may result in degeneration. One can thus understand why, owing to the precocious onset of the disease, there are to be found areas of degeneration or absence of other anatomic structures which were developed prior to the disease, such as the optic reflex tract, nuclei of the abducens and optic nerve fibers. This would explain why in a third of all cases the disease began at the same period of life, especially at the time of puberty when the loss of balance of the endocrine glands is so easily acquired. One finds the same parallel in other organic neuroses, namely, muscular pseudohypertrophy and spinocerebellar aplasia (*morbis Friedreichi*). One might object that in some cases of spinal spastic paralysis the mentality was almost normal, but it is known that even in pure types of Little's syndrome the mental symptoms may be slight. This harmonizes with the fact that aplasia of the Rolandic cortical cells is sufficient to produce Little's syndrome; on the other

hand, pupillary disturbances, convergent strabismus and congenital psychic arrest are met with rather frequently in this disease.

It is only the cases of Artigas and of Friedmann, in which the patients were more or less permanently cured under specific treatment, that can be interpreted as the result of strictly medullary localization, caused by syphilitic and not parasyphilitic processes, as one must presume to be the case in all of the other patients.

I feel justified in making the foregoing statements because, instead of speaking (according to Nonne) of "spinal spastic paralysis with or without cerebral symptoms," I prefer to call the syndrome "spastic paralysis of spinal type." This follows the tendency of modern neuropathology of placing in the cerebrospinal class certain disturbances hitherto classed as purely spinal. This classification is, up to the present day, applied to tabes, amyotrophic lateral sclerosis and spinocerebellar aplasia (Friedreich and Pierre Marie); at present it should be applied also to the spastic-spinal syndromes on a heredosyphilitic basis.

THE PATHOGENESIS OF EPILEPSY FROM THE HISTORICAL STANDPOINT,

WITH A REPORT OF AN ORGANIC CASE *

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Epilepsy, or the sudden loss of consciousness accompanied by convulsions, has always fascinated and baffled the observer. That primitive man is intensely interested in the disease is evidenced by numerous and detailed hypotheses of etiology and by still more numerous therapeutic measures. The primitive mind in its anthropomorphic conception of the universe and its phenomena frequently confuses *post hoc* with *propter hoc*.

Not only in epilepsy, but "in any painful illness, especially when the sick man is tossing and shaking in fever, or writhing in convulsions on the ground, or when in delirium or delusion, he no longer thinks his own thoughts or speaks with his own voice, but with distorted features and strange unearthly tones breaks into wild raving, the explanation which naturally suggests itself is that another spirit has entered into or possessed him. Any one who watches the symptoms of a hysterical-epileptic patient, or a maniac, will see how naturally in the infancy of medical science demoniacal possession came to be the accepted theory of disease, and the exorcism or expulsion of these demons the ordinary method of treatment."¹

Some primitive tribes hold that the evil spirits or demons, who enter a person and cause the epileptic phenomena, live ordinarily in animals (animal spirits) or that they may come from the realm beyond the natural. Other tribes think that the disease is due to temporary escape of the frightened soul from the body,² and when one observes an epileptic child with distorted features and outstretched hands issue a sudden cry as if frightened and fall unconscious to the ground, this primitive idea of a frightened soul escaping from the body does not

* From the Laboratory of the Massachusetts State Psychiatric Institute, Boston.

1. Tylor, E. B.: *Anthropology*, New York, 1909, pp. 353 and 354.

2. Bartels, M.: *Die Medizin der Naturvölker*, Leipzig, 1893, pp. 15, 22, 23, 38, 42 and 213.

appear so absurd. "According to the Belqula or Bella Coola Indians of British Columbia the soul dwells in the nape of the neck and resembles a bird enclosed in an egg. If the shell breaks and the soul flies away, the man must die. If he swoons or becomes crazed, it is because his soul has flown away without breaking its shell."³

But even more civilized people hold superstitious ideas about epilepsy. Thus in upper Bavaria it was formerly thought that epileptic persons were tortured by the spirit of some departed, greedy for a living soul.⁴ It is notable that many primitive races assign natural causes to the disease. In the Celebes epilepsy is regarded as hereditary.

The older civilized races have penetrated into the probable etiology of epilepsy. Some of them, however, still hold to the idea of demoniacal possession. "Epilepsy, while not mentioned in the Old Testament, is often alluded to in the New. The Greek designation *Seleniazomenoi* (literally 'moonstruck') owes its origin to the idea that the disease was due to the moon. In the new Testament period this illness was attributed to demoniac possession though Matthew usually distinguishes between the possessed and the lunatic."⁵ Here a new cause, a natural cause, is introduced, namely, the influence of the moon. That this celestial body exerts a pathogenic influence on the human constitution is held in certain districts of Latwija, known to my personal experience. The source of this idea is probably the Bible. It may be that the moon, especially the full moon, has a definite effect on certain neurotic persons, as has been recently shown by J. Sadger.⁶

The Hindus assign psychologic causes to epilepsy. "Epilepsy, Apasmara, literally forgetting, arises in the channels of the heart, especially by worry, anger, desire, fright, joy, and other emotional excitements, and manifests itself by contractions of eyebrows, mouth, clouding of consciousness, fainting, dizziness, sweating, flatulence, weakness, and other manifestations."⁷ It is significant that these mental factors have been recently reemphasized.⁸

But it was left to the Greek mind to tear down supernatural causes and point to the head (the brain) as the seat of the lesion causing

3. Frasier, J. C.: *The Golden Bough. A Study in Magic and Religion*, Ed. 3; *Taboo and the Perils of the Soul*, New York, Macmillan Co., 1911, Pt. 2, p. 34.

4. Hoffer, M.: *Volkmedizin und Aberglaube in Oberbayerns Gegenwart und Vergangenheit*, München, 1893.

5. *The New Schaff-Herzog Encyclopedia of Religious Knowledge*, New York 3:447, 1909.

6. Sadger, J.: *Ueber Nachtwandeln und Mondsucht. Eine Medizineschliterarische Studie*, Wien, 1914, pp. 1-171.

7. Jolly, J.. *Medicin*. In "*Grundriss der Indo-Arischen Philologie und Altertumskunde*," Strassburg 3: No. 10, p. 121, 1901.

8. Clark, L. P.: *Clinical Studies in Epilepsy*, *Psychiat. Bull.* 2:21, 1917.

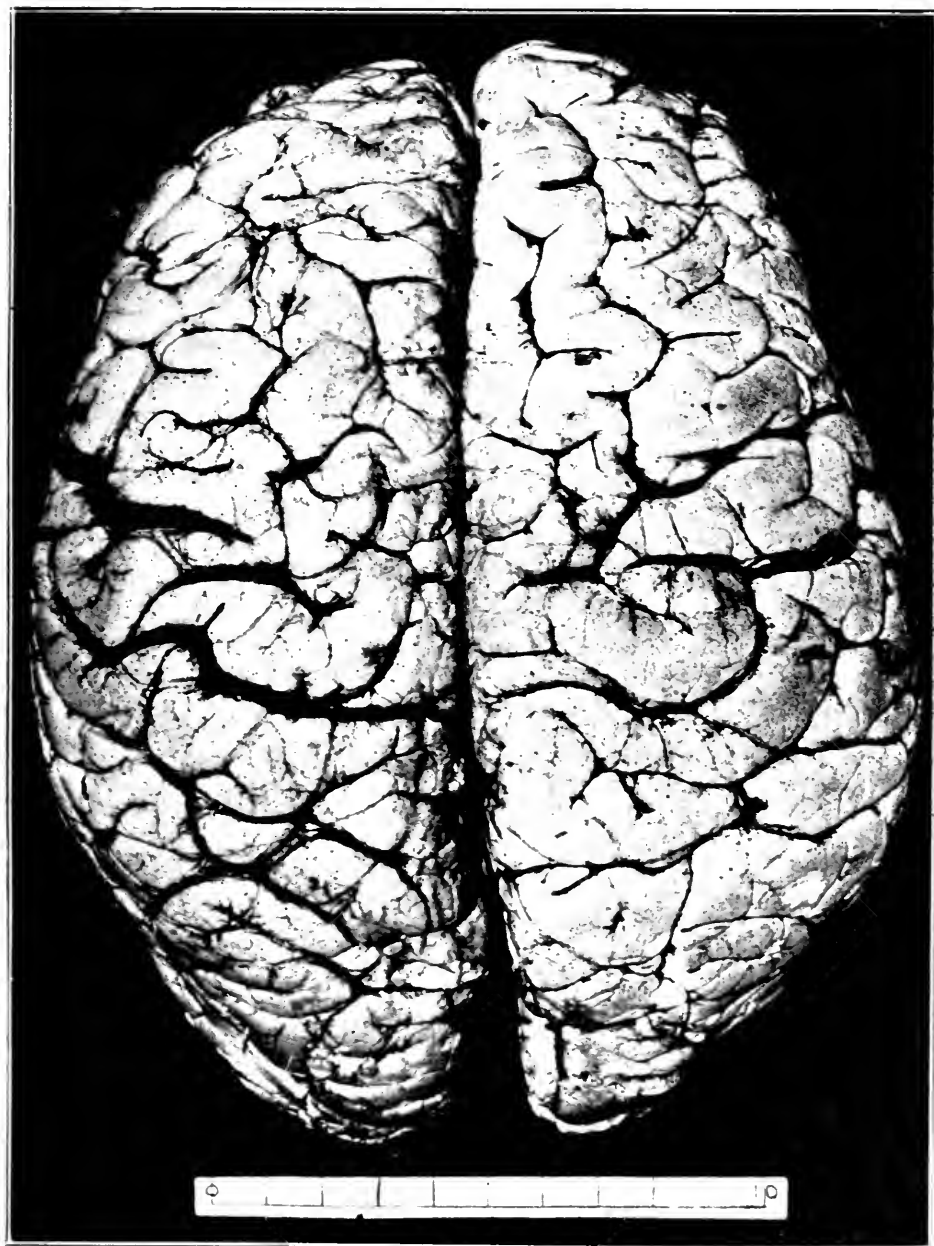


Fig. 1.—Superior surface of stripped brain: enlargement of left frontal lobe.

epilepsy. Here medicine became entirely separated from metaphysical speculation and philosophy. The clearest exposition of the etiology of epilepsy is that of Hippocrates:⁹

"For, if they (epileptic patients) imitate a goat, or grind their teeth, or if their right side be convulsed, they say that the mother of the gods (Cybele) is the cause. If they speak in a sharper, shriller tone, they liken this state to a horse and say that Poseidon is the cause . . . But if foam be emitted by the mouth and the patient kick with his feet, Ares (Mars) gets the blame. But terrors which happen during the night, and fevers, and delirium, and jumpings out of bed, and frightful apparitions, and fleeing away—all these they hold to be the plots of Hecate, and the invasions of the Heroes, and use purifications and incantations, and as it appears to me, make the divinity to be most wicked and impious." "Its origin is hereditary, like that of other diseases." ". . . the brain is the cause of this affection, as it is of other very great diseases, and in what manner and from what cause it is formed, I will now plainly declare." "If the secretion (melting) from the whole brain be greater than natural, the person, when he grows up, will have his head diseased, and full of noises, and will neither be able to endure sun nor cold." "Or, if the depuration do not take, but it (secretion?) accumulates in the brain, it necessarily becomes phlegmatic." "The man becomes speechless when the phlegm, suddenly descending into veins, shuts out the air, and does not admit it either to the brain or to the vena cava, or to the ventricles, but interrupts the inspiration." In other words, "when the veins are excluded from the air by the phlegm and do not receive it, man loses his speech and intellect, and the hands become powerless, and are contracted, the blood stopping and not being diffused as it was wont."

"All these symptoms he endures when the cold phlegm passes into the warm blood, for it congeals and stops the blood." "For the brain becomes more humid than natural, and is inundated with phlegm, so that the defluxions become more frequent, and the phlegm can no longer be excreted, nor the brain be dried up, but it becomes wet and humid. This you may ascertain in particular, from beasts of the flock which are seized with this disease, and more especially goats, for they are most frequently attacked with it. If you cut open the head, you will find the brain humid, full of sweat, and having a bad smell (hydatid). And in this way truly you may see that it is not God that injures the body, but disease, and so it is with man. For when the disease has prevailed for a length of time, it is no longer curable, as the brain is corroded by the phlegm, and melted, and what is melted down becomes water, and surrounds the brain externally, and overflows it; wherefore they are more frequently and readily seized with the disease."

In the Middle Ages the etiologic explanation of the Old and New Testaments was accepted. Epileptic persons were looked upon as possessed by the devil. Only the Byzantine and Arabian physicians upheld the Greek tradition in medicine. A detailed discussion of epilepsy is found in the works of Alexander of Tralles¹⁰ (525-605 A.D.)

9. Hippocrates: *The Genuine Works of Hippocrates*, Trans. from the Greek by Francis Adams, New York, 2:338-339, 1886.

10. Von Tralles, Alexander: *Ueber die Epilepsie in "Original-Text und Uebersetzung,"* vol. 2, edited by Puschmann, Wien, 1878, pp. 534-535.

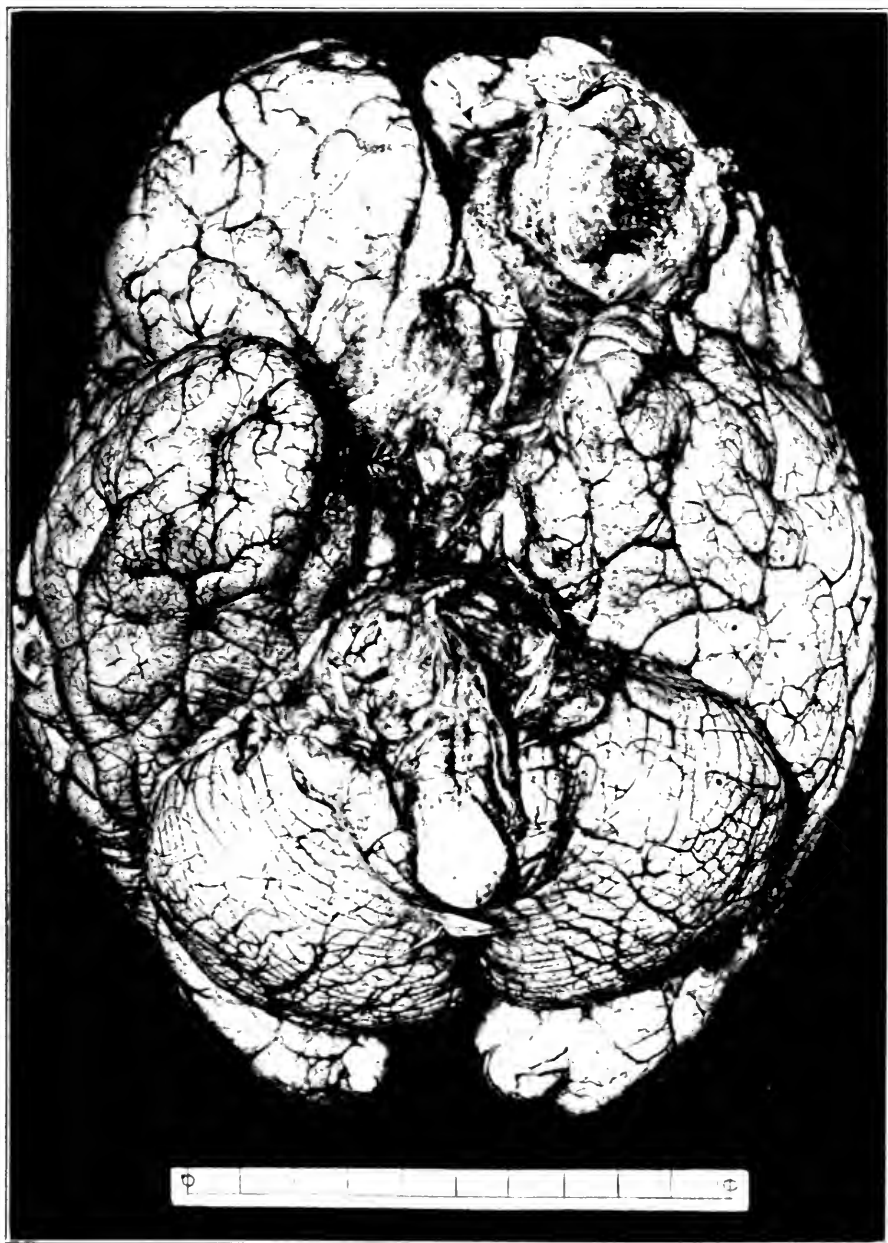


Fig. 2.—Basal surface of unstripped brain. The cerebellum fits closely about the medulla. The left frontal lobe shows an umbilicated tumor mass. The left olfactory lobe has been destroyed.

"The disease has its seat in the head, where the sensation and motion arise. That the head is the affected part is proven by the condition of the patient during the epileptic convulsion. They can not hear, see, or perceive in general, nor remember, but lie devoid of all sensation, and do not differ in anything from the dead; wherefore the disease is also called *epilepsia*, because the perception of the patient is extinguished and suppressed. Others call it the sacred disease, because the brain is regarded as something sacred and precious, while some call it the disease of Heracles, because it appears suddenly and is difficult to remove." "Epilepsy can arise in three different ways: It can have its origin directly from the head, or from the stomach, or from other parts of the body, and transplant the disease contained in them to the head."

Nothing of importance was added to the explanation of epilepsy until the nineteenth century, when Brown-Séquard experimentally produced epileptic convulsions in 1869-1870.¹¹

But more important is the work of John Hughlings Jackson (1834-1911)¹² who described unilateral convulsions or focal epilepsy (1875) and originated the doctrine of levels in the central nervous system. This three-level doctrine and, in particular, its application to epilepsy, has been excellently discussed by Elmer E. Southard,¹³ to whose article I refer those interested in the subject. The ideas of Jackson stimulated investigation of the pathogenesis of epilepsy. The muscles, the peripheral and sympathetic nervous system, and the bulbospinal apparatus were searched, but they threw comparatively little light on the mechanism of epilepsy. The same is true of the body at large and its fluids.¹³

"It is within the range of possibility that a kind of epilepsy might be produced by a poison acting directly on muscular substance (for convulsions) and on central nervous tissues (for unconsciousness). It is possible that twitchings in the death agony are so produced. But the march of convulsions in ordinary epilepsy suggests a higher origin."¹³

There is no doubt that epileptic convulsions, general as well as partial, arise at least mainly in the cerebral cortex, particularly in the electrically excitable areas.¹⁴ "A paralyzing lesion destroying part of the motor is less likely to be followed by convulsion than one in the vicinity of the center, which slightly damages its structure and deranges its functions."¹⁵

11. Livon, C. Cobaye: In C. Richet: *Dictionaire de Physiologie*, Paris **3**:926, 1898; Alford, S. B.: *Brown-Séquard's Epilepsy in Guinea-Pigs*, Boston M. & S. J. **165**:635-643, 1911.

12. Jackson, J. Hughlings: On the Evolution and Dissolution of the Nervous System, *Lancet* **1**:555-558, 649-652, 739-744, 1884.

13. Southard, E. E.: On the Mechanism of Gliosis in Acquired Epilepsy, *Am. J. Insanity* **64**:608, 610, 611, 1908.

14. Lewandowsky, M.: Die Funktionen des Zentralen Nervensystems, in "Handbuch der Neurologie: Allgemeine Neurologie" **1**: Pt. 2, 743-749.

15. Gowers, W. R.: *Epilepsy and Other Chronic Convulsive Diseases*, London, 1901, p. 160.

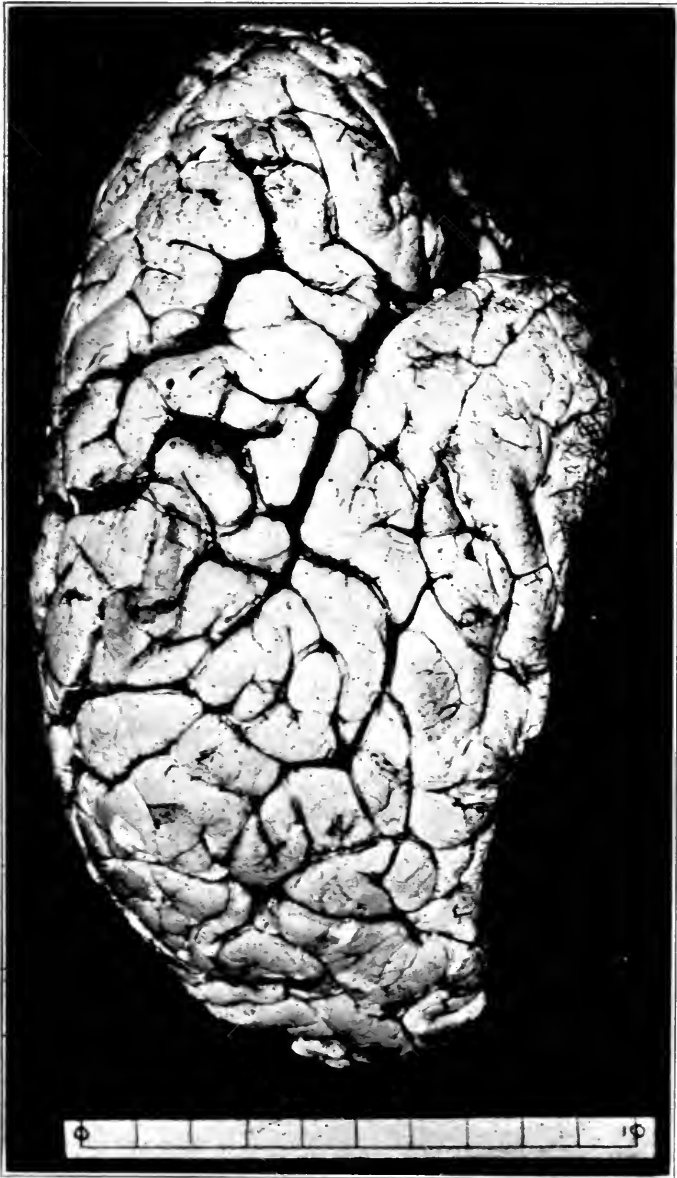


Fig. 3.—Right lateral surface of stripped cerebral hemisphere.

It is unnecessary to discuss all the organic conditions assigned to cause epilepsy. Brain tumors, however, form a rather interesting group. The following case in which the symptoms were those of idiopathic epilepsy, and in which necropsy examination revealed a frontal lobe tumor, belongs in this group.

History.—R. P., a woman, aged 44 years, a widow, a weaver, was admitted to the Monson State Hospital Nov. 11, 1913, at the age of 38 years. The patient stated that her epileptic attacks began four years ago. She thought that she had had two or three fits a week at the beginning but now she had a regular attack once a month "with little ones that don't amount to anything" in between. She was subject to the usual instability before the attacks and to a state of mental confusion following them. She appeared to be in a fair condition of health. No family history was obtainable.

She went to school until the age of 13 years. Several years later she went to work in a mill as a weaver, and married at the age of about 20. She had six children; two boys and two girls were living, one child was stillborn and another died in infancy. The eldest child was about 12 or 13; the patient did not know their whereabouts. Her husband died three or four years ago. She had measles in childhood. She had been alcoholic since youth, drank several bottles of beer and several drams of hard liquor every few days; became intoxicated once a week. Formerly she took snuff. She acknowledged that the last child was illegitimate. Menstruation was regular. She denied venereal disease.

Epileptic convulsions began four years ago. She had had seizures of both the grand and petit mal type. The auræ were sometimes gastric and at other times she had a sensation of dizziness.

Physical Examination on Admission.—This revealed nothing of importance.

Mental Examination.—Her memory both for recent and past events was poor. She was correctly orientated for time, place and person. Insight was good. She realized that her last child was illegitimate and expressed shame; she freely acknowledged that she used alcohol and considered this the cause of her epilepsy. She declared with all earnestness that she would never drink any more and felt that she could withstand any temptation.

During her stay in the hospital the patient had spells in series, usually at night, occurring about the time of her menstrual period; these were grand mal attacks with a few petit mal attacks and were followed by severe headaches and superficial soreness of the scalp. Nose and throat treatment, electric baths and sodium bromid did not result in any lasting beneficial effect. During attacks the patient occasionally injured herself. During the summer of 1917 the patient began to show progressive mental deterioration which by January, 1919, had become marked. The attacks became more frequent during January and February. The patient died Feb. 26, 1919, in the afternoon, following an attack in the morning.

Necropsy Examination.—Twenty-two hours postmortem. Only the essential findings of a very detailed examination were reported. Body that of a fairly well built, well developed and well nourished white female. Uterus retroposed. Cystic organ of Rosenmueller on right. Left Fallopian tube injected. Right lung has rudimentary middle lobe. Abdominal and pelvic viscera were negative. Head: The inner surface of the calvarium showed elevations and depressions and was unusually dry. The dura was tightly stretched over the brain

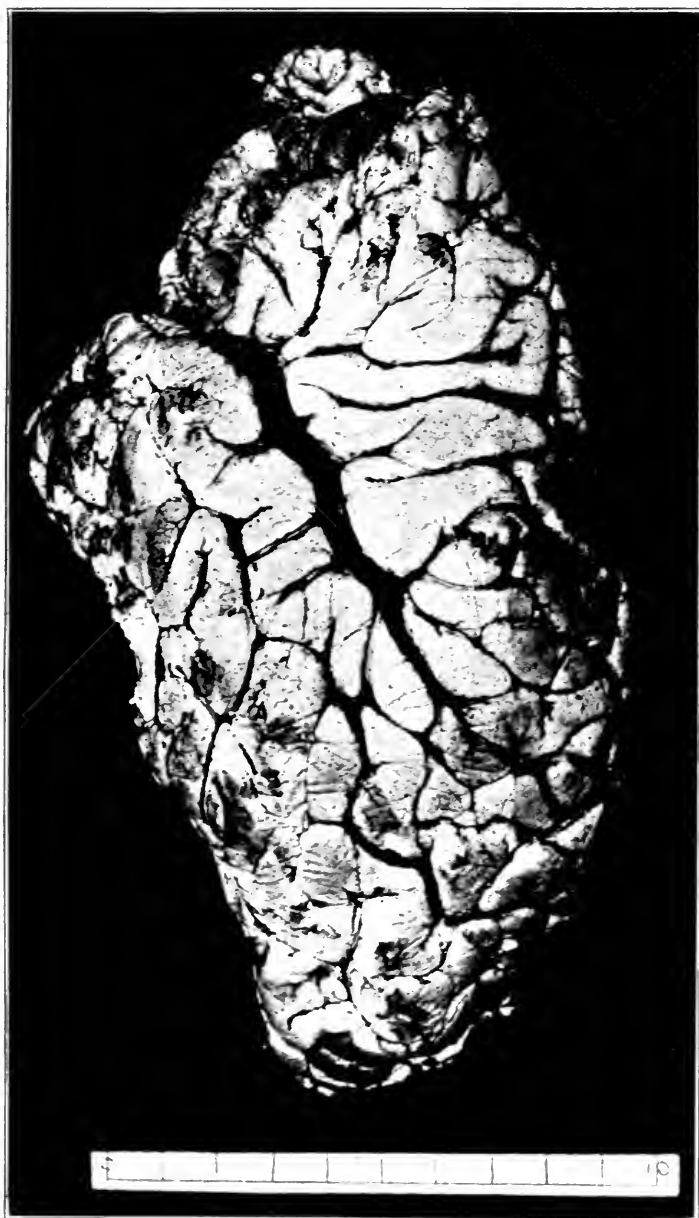


Fig. 4.—Left lateral surface of stripped cerebral hemispheres. Distortion and partial destruction of the frontal pole.

and convolutions could be seen through it. Over the midfrontal region fluctuation was palpated. The frontal lobe was adherent to the orbital plate of the frontal bone. The base of the brain showed an umbilicated, yellowish gray body, measuring 3.8 cm. in diameter in the left frontal lobe. The edges were granular and it was surrounded by gelatinous and softened brain tissue. The left olfactory bulb was destroyed by this growth. The tips of the lobus pyramiformis were markedly lipped, especially on the left. The middle cerebral arteries were more sclerosed than the basilar. The temporal poles were uneven, the left being larger. Abruptly at the Sylvian fissure the brain tissue was soft and thin. Fluid withdrawn from the free edge of the body was yellow. Fluid from the third ventricle was clear. The superior surface of the brain was dry and glossy, with here and there a diffused blood content in the veins and a pressed out appearance of the veins. The medulla and pons were broad. The cerebellum fitted closely about the medulla. The orbital surface of the left frontal bone was elevated in a circular manner and to this surface the brain substance and portions of growth were attached. The bone itself in the central portion was eroded, giving this part of the bone a "Tam o' Shanter" appearance. The left optic disk showed some edema; the right was choked. The brain weighed 1,520 gms. Tigge's formula $8 \times 152: 1216$. The gain was 304 gm. The pituitary gland was soft, especially the posterior lobe.

Cause of Death.—Hydrocephalus was the cause of death.

Microscopic Diagnosis.—Psammoma in the left frontal lobe was diagnosed microscopically.

Cerebrospinal Fluid.—Cerebrospinal fluid findings were: cells, 36 (small lymphocytes 27, endothelial 2, polymorphonuclear 7); globulin +++; albumin +++; colloidal gold curve 0044553322; Wassermann reaction, unsatisfactory.

PSYCHIC DISTURBANCES CAUSED BY CEREBRAL TUMORS

Statistical compilations show that patients with cerebral tumors exhibit psychic disturbances in at least two thirds of the cases. This depends on the character and location of the tumor: bulb, one-fourth; cerebellum, one-third; hypophysis, two thirds; corpus callosum, all without exception (Bechterow,¹⁶ Dercum¹⁷).

Among these psychic disturbances fits of unconsciousness with convulsions are frequent in cerebral tumors apart from cortical epilepsy, the presence of which depends on the definite location of the tumor. Attacks of genuine epilepsy, which may manifest itself at any stage of the disease, may be the precursor."¹⁸ "In rare cases, general epileptic convulsions may for a long time be the only symptom produced by an intracranial tumor."¹⁹ It may be impossible to distinguish them

16. Bechterew, W.: Troubles psychique dans les maladies nerveuses organiques, in "Traité international de psychologie pathologique," edited by A. Marie, Paris 2:78-154, 1911.

17. Dercum, F. X.: A Clinical Manual of Mental Diseases, Ed. 2, 1917, p. 315.

18. Oppenheim, J.: Lehrbuch der Nervenkrankheiten, Ed. 6, Berlin 2:1166, 1913.

19. Bramwell, B.: Intracranial Tumors, in Allbutt and Rolleston: System of Medicine, London 8:226-290, 1911.



Fig. 5.—Mesial surface of stripped left cerebral hemisphere. The frontal lobe has been distorted and partially destroyed.

from ordinary idiopathic epilepsy until double optic neuritis, headache or other signs appear.¹⁹ But it is doubtful whether epilepsy may be regarded as the first symptom, since often one may assume that a brain so organized is especially suitable soil for neoplasm.¹⁸



Figs. 6 and 7.—Frontal sections through the frontal lobes showing the tumor; marked tissue destruction of the left lobe.

With the possible exception of tumors in the corpus callosum, frontal lobe tumors show psychic disturbances more frequently than others (Schuster,²⁰ Duret,²¹ Bechterew,¹⁶ Dercum¹⁷). But mental

20. Schuster, P.: *Psychische Störungen bei Hirntumoren*, Stuttgart, 1902,



Figs. 8 and 9.—Frontal sections showing the psammoma. There is considerable distortion of the left hemisphere and encroachment on the right.

symptoms, regarded by some as characteristic of frontal tumor, are also seen, though less frequently, in tumors elsewhere. Negative cases, as regards mental symptoms, are observed even in bilateral frontal lesions.² There is no doubt that in the animal kingdom intellectual capacity runs parallel with development of the frontal lobes. However, the well developed frontal lobe is not characteristic of primates alone. Ungulates show well developed frontal lobes which relatively are hardly smaller than that of primates. Monakow's measurements in horses, cattle and goats show the frontal lobes to be 25 per cent. of

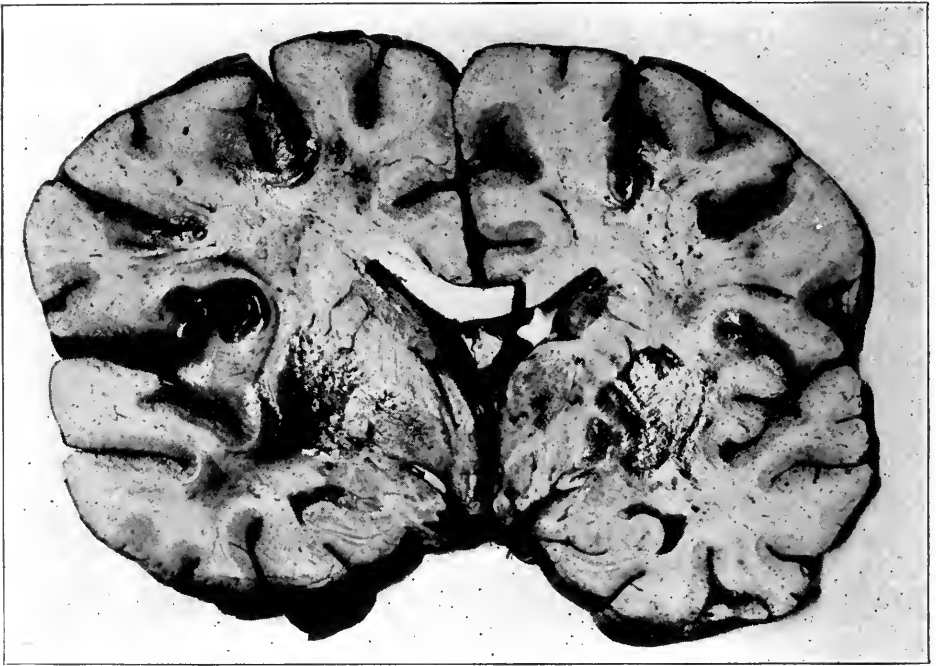


Fig. 10.—Frontal section passing through the thalami. This still shows the effects of the tumor.

the total brain volume; besides the lobe does not taper by any means. Further, this frontal cortex shows quite a number of well-formed convolutions. Despite all this there is no definite mental superiority of the ungulates over the carnivora. The well developed frontal lobe in ungulates is probably a counterbalance to the poorly developed regio sigmoidea (cortical representation of the extremities²²).

But even in animals as high as the monkeys the function of the frontal lobes cannot be considered as definitely established. Horsley

22. Monakow, C.: *Die Lokalisation im Grosshirn*, Wiesbaden, 1914, pp. 883-885.

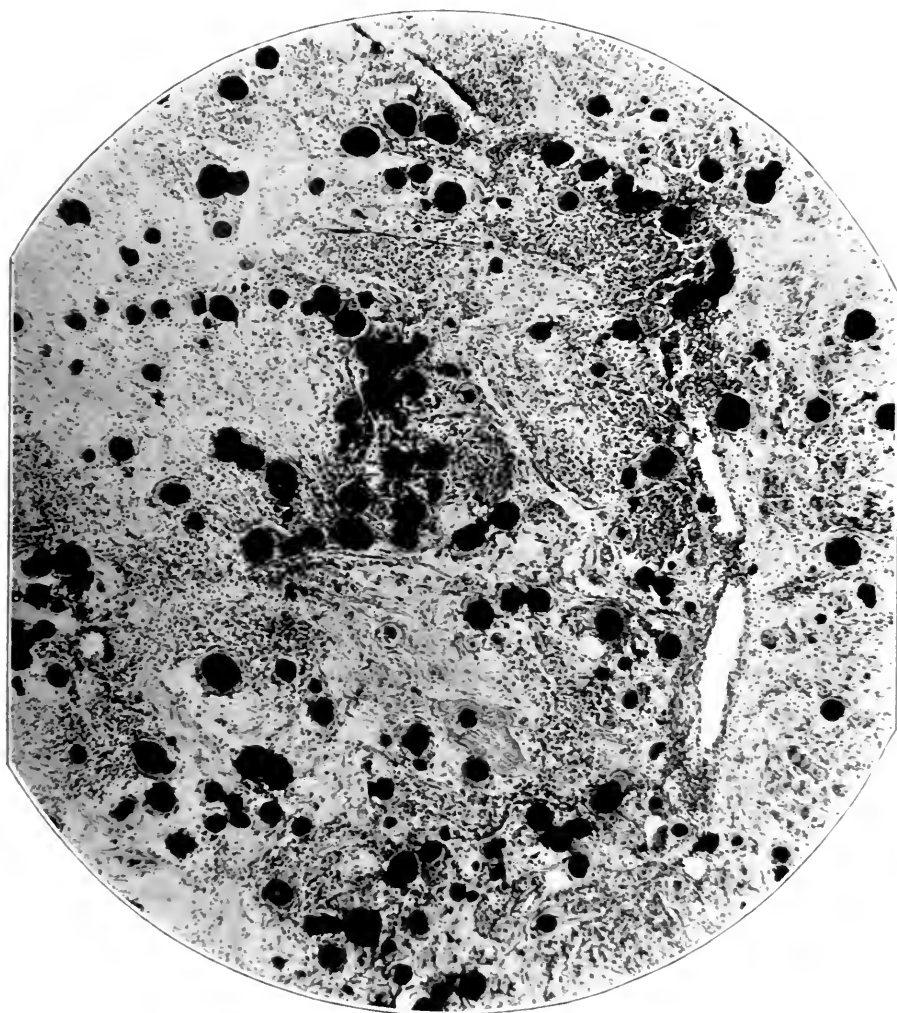


Fig. 11.—Microscopic section of the tumor, psammoma, showing numerous whorls composed of large flat cells arranged concentrically. In some whorls calcium is deposited and forms concretions.

and Schäfer²³ report an experiment on a monkey which, following extirpation of both frontal lobes, was kept alive for almost three months; during this period the animal showed no disturbance of the intellect. Later Graham Brown and Sherrington²⁴ showed that in a chimpanzee destruction of the motor cortex does not cause permanent paralysis. In their experiment the left arm center was removed with resulting paralysis on the right. Complete recovery took place in four and a half months. Regeneration of the area was excluded by the fact that six and a half months after the first operation the arm cortex was electrically inexcitable. The taking over of the movements by the postcentral electrically inexcitable convolution was excluded by removing this convolution two months after the second operation (Brown and Sherrington,²⁴ Bayliss²⁵).

In spite of all this somewhat baffling experimental work on the frontal cortex, the frontal lobes may be looked upon as exerting a "psychoregulatory function."²⁶ In the left cerebral hemisphere the motor centers predominate over the right, while the sensory cortex is better developed in the right hemisphere in right-handed persons. These functional differences are only quantitative, but in regard to higher psychic functions, they are qualitative.²⁶

The mental disturbances of frontal lobe tumors are "comparable to symptoms of paresis: indifference, unpunctuality, mental enfeeblement, loss of memory and power of attention, change in disposition with more or less marked irritability or taciturnity or obstinacy or jocularity, etc., rambling speech, lack of realization of illness, change in general conduct of life, habits of untidiness."²⁷

The general symptoms of intracranial tumors are produced mainly by retention and increased tension of the cerebrospinal fluid.²⁸ Anatomically the effects of pressure are seen in cerebral anemia, distur-

23. Horsley, V., and Schäfer, E. A.: A Record of Experiment Upon the Functions of the Cerebral Cortex, *Philosoph. Trans.*, 1888, p. 179 (cited by Monakow, p. 888).

24. Brown, T. G., and Sherrington, C. S.: Note on the Functions of the Cortex Cerebri, *Proc. Physiol. Soc., J. Physiol.* **46**:22, 1913; Brown, T. G.: Studies in the Physiology of the Nervous System, *Quart. J. Exper. Physiol.* **10**:103-143, 1916; Sherrington, C. S.: Stimulation of the Motor Cortex in a Monkey Subject to Epileptiform Seizures, *Brain* **41**: Pt. 1, 48, 1918.

25. Bayliss, W. M.: *Principles of Physiology*, Ed. 2, London, 1918, p. 480.

26. Bechterew, W.: *Die Funktionen der Nervenzentra*, Jena **3**:2010, 1911.

27. Cushing, Harvey: Tumors of the Brain and Meninges, in Osler and McCrae: *Modern Medicine*, Ed. 2 **5**:308-350, 1915.

28. Bruns, L.: *Die Geschwülste des Nervensystems*, Ed. 2, Berlin, 1908, p. 73.

bances of lymph circulation, edema and other diffuse histologic injuries of the cerebral cortex (degeneration of ganglion cells with increased satellitosis, disappearance of longitudinal fibers, etc.), and meningeal changes.²⁹ Meynert³⁰ speaks of a "functional choking of cortical function (Leistungen) running parallel with the mechanical pressure," while Dupré³¹ emphasizes the toxic besides the mechanical factors in the symptomatology of cerebral tumor.

Kraepelin³² thinks that psychic disturbances in tumors of the hemispheres are due to destruction of brain substance, to pressure impairing cerebral circulation and possibly to traction and displacement of the tissue with accompanying injuries. Further, one may under certain conditions consider absorption of decomposition products. Finally, as pointed out by Redlich,²⁹ in brain tumors one has to deal with the possibility that besides the new growths there are finer changes in other regions of the brain which may easily be overlooked.

But all these findings, even the most recent ones, such as gliosis and similar changes in the cerebral cortex of epileptic patients, add little to the understanding of the disease. They only point to organic changes underlying epilepsy.³³ In the present state of our knowledge it is impossible to postulate an anatomic epilepsy,³⁴ since there are no changes in the central nervous system pathognomonic of epilepsy (Voisin,³⁵ Tramer³⁶).

SUMMARY

This was apparently a case of idiopathic epilepsy which postmortem examination proved to be a psammoma of the left frontal lobe, with hydrocephalus. Epilepsy among primitive people is thought to be due to the possession of demons, animal spirits, escape of soul from the

29. Redlich, E.: *Die Psychosen bei Gehirnerkrankungen*. Aschaffenburg: Handbuch der Psychiatrie, Spez. Teil III, Bd. II, 1912, p. 335.

30. Meynert: *Klinische Vorträge über Psychiatrie*, Wien, 1890, p. 267.

31. Dupré, E.: *Psychopathies organiques*, in Ballet, G.: "Traité de pathologie mentale," Paris 6:1184, 1903.

32. Kraepelin, E.: *Psychiatrie*, Ed. 8, Leipzig 2: Pt. 1, 36, 1910.

33. Strümpell, A.: *Lehrbuch der Speziellen Pathologie und Therapie der Inneren Krankheiten*, Ed. 19, Leipzig 2:741, 1914.

34. Binswanger, O.: *Die Epilepsie*, Ed. 2, Wien und Leipzig, 1913, p. 357.

35. Voisin, J.: *L'épilepsie*, Paris, 1897, p. 293.

36. Tramer, M.: *Untersuchungen sur pathologischen Anatomie des Zentralnervensystems bei der Epilepsie*, Schweiz. Arch. f. Neurol. u. Psychiat. 2:202, 1918.

body, and heredity. Civilized races hold that the disease is due to demoniacal possession, the influence of the moon, emotional excitement and lesions of the brain.

Epilepsy is due to various causes, brain tumor occasionally causing this disease. Anatomically there are no changes in the central nervous system pathognomonic of epilepsy.

GLOBAL APHASIA AND BILATERAL APRAXIA DUE TO AN ENDOTHELIOMA COMPRESSING THE GYRUS SUPRAMARGINALIS *

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Tumors involving the cerebral cortex, provided they are small and well limited, may furnish valuable information on cerebral localization.

In Dr. Cushing's clinic I have had the opportunity to observe a patient in whom a small endothelioma compressing the left gyrus supramarginalis had produced global aphasia and bilateral ideomotor apraxia, which disappeared after the removal of the tumor. The essential facts in the history are given in the case report.

History.—Mr. C. (Surgical No. 13344), aged 60, right-handed, in November, 1919, while driving his car experienced sudden numbness in the right arm and hand, with loss of speech. After driving ten miles in this condition, he was able to say only "Yes" and "I don't know." He soon had a generalized convulsion. All symptoms had disappeared by the following day. During 1920 five similar attacks occurred. Between the attacks the patient was apparently normal and continued to perform his duties as treasurer of a bank.

On Oct. 14, 1920, after the usual sensory prodromes, the patient had another generalized convulsion which was soon followed by two other severe attacks. When brought to the hospital the same day the right arm was constantly twitching; he could not understand the simplest orders and could say only "I don't know." The next day the twitchings had disappeared.

Examination.—The general physical examination was negative. Neurologic examination showed slight obscuration of the temporal margin of the left disk without swelling; slight paresis of the right arm which, however, he was continually moving and which showed astereognosis. The radial reflex was exaggerated on the right. Other reflexes and the plantar reflexes were normal. The patient was good humored and made every effort to cooperate.

There was global aphasia. Incomprehension of speech was complete. Sometimes a word was recognized and the patient guessed the sense of the order. No object could be named. Speech was limited to "It is funny" and "I don't know." Alexia was present, and he could write nothing except his name correctly. There was bilateral ideomotor apraxia. When offered a familiar object (matchbox, purse, etc.) he took it with his right hand (with the left only on insistence). He would hide it in his bed, take it out and regard it with perplexity, put it on his head, or execute most absurd movements with it; for example, quick alternative flexion and extension of the elbow. When offered a cigaret and a box of matches together, he put the cigaret in his mouth but executed with the matchbox the same absurd movements. The same confusion, though to a less degree, was apparent when using familiar objects like a fork and spoon in eating. On the succeeding days the same apraxia persisted. The patient frequently turned the head and eyes to the right as though subject to visual hallucinations.

* From the Surgical Service of the Peter Bent Brigham Hospital.

Diagnosis.—The diagnosis of postcentral tumor was made, based on the sensory features of the attacks, the slight paresis of the arm, the global aphasia and the apraxia (Marie and Foix's syndrome of the gyrus supramarginalis).

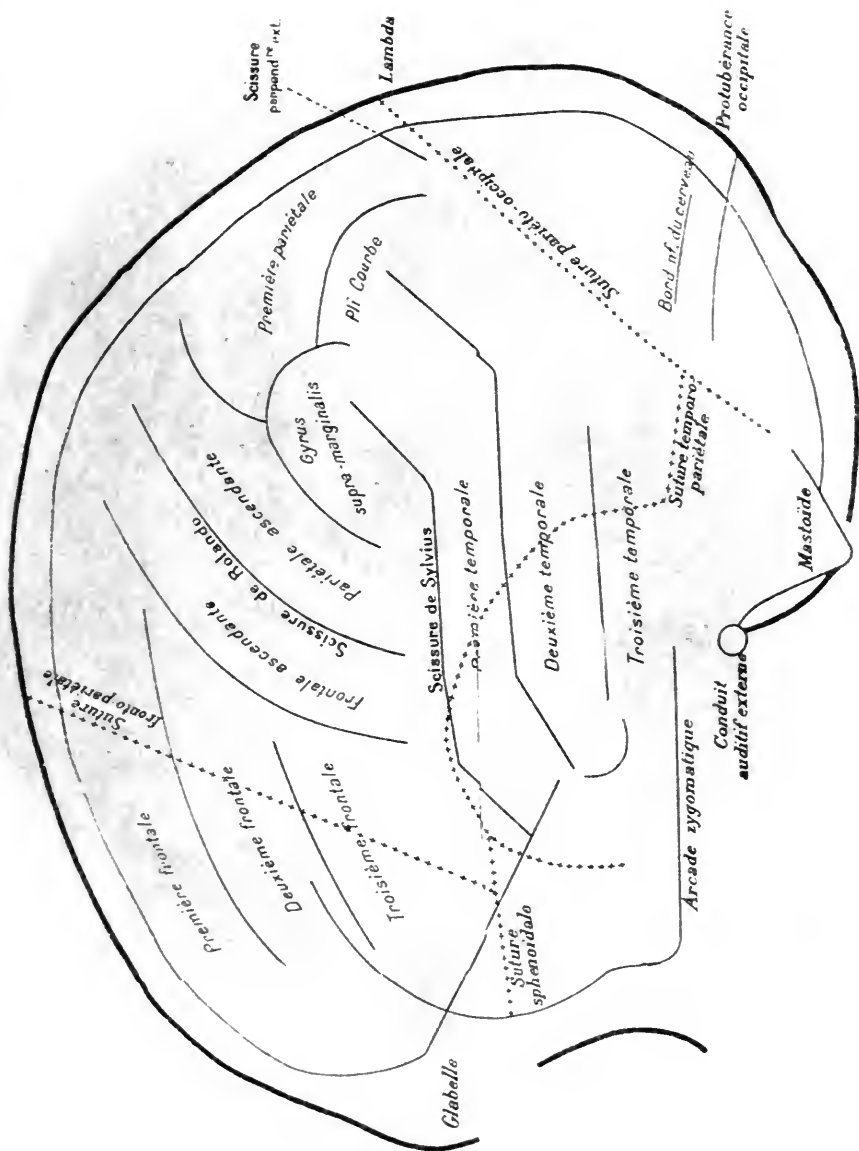
Treatment and Course.—On October 19, Dr. Cushing enucleated from the left gyrus supramarginalis an endothelioma measuring 4 by 3.5 cm. (Fig. 1). Its position was easily recognized by the situation of the Sylvian fissure and vein. This was subsequently verified on the roentgenogram by the use of Marie's scheme (Fig. 2). The rest of the cortex seemed quite normal and the brain was under no increase of tension.

The operation, though a simple one, was unfortunately complicated by a postoperative clot which formed in the cavity left by the tumor and



Fig. 1.—Lateral view of the tumor showing dural attachment below.

necessitated reelevation of the flap later in the day. This accident unquestionably produced some local damage, for on the following day there was moderate hemiparesis predominating in the upper limb, without a Babinski sign. The same ideomotor apraxia persisted as before (amorphism of the movements suggested). This naturally could be observed only in the left hand owing to the right paralysis. Power of speech was recovered gradually and was characterized by marked dysarthria and some apraxia (perseveration). Incomplete right hemianopsia was found. Soon the apraxia took a milder character and persisted for more than a month. He had unquestionable comprehension of the act to do, as proved by his mimicry (e. g., his perplexity, Figs. 3 and 4), and his perseverance, was not agnostic, but showed



12

... This accident
... the following day
... in the upper limb.
... perceived as
... This naturally could
... Power
... marked
... light beam
... milder character and
... earnest confidence
... his perplexity.
... agnostic, but showed



Fig. 2. Postoperative roentgenogram showing outline of bone flap with dilated diploetic vessels. The circle of silver clips on the margin of the excised dura is shown, and at the center point "X" represents a burr opening in the flap which was the central point of the tumor in which a small bony exostosis was present on the inner surface of the skull. (Compare with Marie's *schizma* indicating the radiographic projection of the cranial sutures and cerebral convolutions.)



Figs. 3 and 4.—Degrees of perplexity of the patient after long unsuccessful trials to light the candle. He had previously been unable to light his cigaret which was finally lighted for him.

either omission of one of the essential elements of the act or its replacement by an inappropriate one (striking the candle against the box of matches, etc., Fig. 5), without special clumsiness of the individual movements. Occasionally he would be able to perform a given test at once, as it were automatically, but when he had failed at the beginning he could rarely accomplish the test (perseveration). The expressive gestures were correct. His mental condition was excellent, as proved by his attentiveness, efforts to reeducate his speech, judgment, sense of humor and entire behavior.

Recovery was progressive and when seen for the last time, three months after the operation, he had still definite dysarthria but only



Fig. 5.—Patient endeavoring to strike the unlighted match on the candle.

slight aphasia (difficulty in writing words containing numerous vowels, but he could read perfectly). There was no further trace of the apraxia. He was in good general condition, could use his right arm perfectly and could walk several miles.

This case seems to fulfil all the conditions required for a brain tumor to furnish valuable evidence regarding localization; namely, a small and enucleable tumor and minimized pressure conditions, as shown by the absence of choked disk or distant symptoms. In three other of Dr. Cushing's patients occasion was offered to appreciate the

practical value of the localizations given by Marie and Foix: In a case of an encapsulated glioma the size of a large hen's egg situated in the region of the gyrus supramarginalis and angular gyrus there was the same global aphasia with apraxia, both of which disappeared after the removal of the tumor, the aphasia and alexia immediately, the apraxia and the agraphia after about eight days. The day following the operation, the patient, an accountant, could sustain a long conversation, was not only clear but witty, and yet when asked, for example, to light a cigaret forgot to strike the match, but struck it against the cigaret in obstinate and vain trials. He said later he thought the matches were "fake ones." This dissociation, during the recovery, between the aphasic and apraxic symptoms is rather interesting.

In two other cases a small gliomatous cyst of the frontal region had produced the type of aphasia characterized by the intensity of the dysarthria contrasting with the relative conservation of the understanding. This represents the syndrome anarthrique of Marie and Foix, which they showed to be produced by a lesion in the posterior part of the second frontal convolution and the adjacent part of the ascending gyrus.

1. A patient with a tumor of the gyrus supramarginalis showed slight paresis of the right superior limb with marked sensory disturbances, global aphasia, and ideomotor apraxia. These are the elements of the syndrome of the gyrus supramarginalis described by Marie and Foix grounded on their experience with aphasia resulting from war wounds.¹

2. The case agrees with the notion generally admitted of the possible production of bilateral ideomotor apraxia by a lesion at the level of the left gyrus supramarginalis. On account of the small dimensions of the tumor and its superficial situation, compression of the corpus callosum was out of the question. A lesion of the left gyrus supramarginalis has been found responsible for a true bilateral apraxia in thirteen cases of the forty-one with anatomic verification published in 1914 (von Monakow²). Marie and Foix, in their series, found apraxia in only two cases of injury of the supramarginal gyrus. They assume that the lesions may not have been sufficiently deep. But it may be that the apraxic symptoms had been transitory. In the other twenty-eight cases quoted by von Monakow, the lesions, always multiple, often extensive, were found in the most various regions of the brain, even in the thalamus (multiple thromboses or hemorrhage in arteriosclerotic brains, tumors of "unusual dimensions"). The inconclusiveness of the anatomic documents, together with considerations on the multiplicity of elements which must compose the normal handling

(Handlung) have led von Monakow to a rather skeptical opinion on the localizing value of the symptom apraxia. Therefore a case like the present one, and even the one alluded to in the foregoing, because they are real experiments, are most instructive.

1. Marie, P., and Foix, C.: Les aphasies de guerre, *Rev. Neurol.* **24**:53-87, 1917.

2. Von Monakow: *Die Localisation im Grosshirn und der Alban der Funktion durch kortikale Herde*, 1914.

THE REVIVAL OF SPIRITISM

PSYCHOLOGIC FACTORS*

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The only method known to science of extracting information from deceased persons is the discovery of documents.—*Sir Oliver Lodge.*

The reconstruction periods following devastating wars are axiomatically characterized by widespread nervous unrest. The causes and effects of this universal state of "nerves" are too familiar to require mention. One of the most readily understood accompaniments of such a period of readjustment is a recrudescence of all kinds of religious activity and of divers affiliated undertakings.

Of these phenomena perhaps the most striking, because of certain personages involved, is the contemporary wave of spiritism.

In the western world this wave, which has taken form in the spectacular missionary tour of England's foremost spirit-scientist, has flowed across the states from East to West, and is now receding through the provinces from West to East. It promises temporary interest or enthusiasm, to be followed by forgetfulness, awaiting the impetus of a later wave.

Were we to inquire into the earliest antecedents of this movement, we should have to go back to the witches of the Old Testament and the oracles of Greece. Early Greek and Roman writers, from the sixth century B. C. onward, have left descriptions of mysterious happenings among which the modern student of ghost lore would have been entirely at home. The wonderful performances of the twentieth century medium, like many another pseudo-novum, had their prototype and pattern in the early days of our race.

ORIGIN OF MODERN SPIRITISM

But it is not with this ancient story that we are here concerned. Modern spiritism dates from 1848. It had its origin in the humble village of Hydesville, near Rochester, N. Y., in the home of a family named Fox. It may not be impertinent here to suggest parenthetically that as it is one of the avowed functions of the Society for Psychical Research to investigate striking coincidences, the circumstance that this

* Read at the first meeting of the Ontario Neuropsychiatric Association, Kingston, April 28, 1920.

particular family should have borne the name of "Fox" may be worthy of some speculation. Be this as it may, the fact remains that the two Fox sisters, aged 15 and 12 when they began their careers, were the founders of the contemporary school of mediumship.

As the starting point of the modern epoch in the history of credulity, the Hydesville episode merits a few moments' attention. The Fox family, migrating from Rochester to Hydesville, had taken quarters in a house described by a writer of the time as "quiet and respectable, for aught that is stated, and there is no reason to suppose that the timber had been practiced on by elves or the 'good people' who haunt the woods." Shortly after establishing themselves in this house, "they were disturbed after going to bed by various noises—which, however, did not attract much attention, as they supposed them to be made by the rats which do sometimes of themselves have strange doings." At length, after about four months of these experiences, the family one night "determined to go to bed early, so as to get a good night's rest, in spite of all noise, but this was not permitted; the thought then struck Mrs. Fox, whose bed was in the same room with that of her two daughters, that she would question the noise.

"Who makes the noise?" (Silence).

"Is it made by any person living?" (Silence).

"Is it made by one dead?" (Rap).

"By an injured spirit?" (Rap).

"Injured by me or my family?" (Silence).

At this point it was deemed safe and wise to convoke the family and such neighbors as might conveniently be brought to the scene. Further questioning as to the date of the supposed injury, elicited five raps; and when various names were mentioned, there was an accusing rap at the name of a man who had occupied the house about five years previously. Pursuing the interrogation, they were given to understand by affirmative raps that the body of the injured spirit was buried in the cellar and might there be found. They were admonished, however, not to dig for a space of four months. It is recorded that when this period had elapsed some fragments of bone were disinterred in the cellar; but it does not appear to have been ascertained to what animal they belonged, or how long they had lain in the earth.

It will readily be understood that there was no little commotion in Hydesville; and it was not long before the Fox family found it advantageous to leave not only the house, but the village, "as the excitement for or against them was so considerable." The noteworthy circumstance was that in the journeyings of the family, wherever either of the daughters happened to be staying, there the mysterious sounds infallibly occurred; and in due time certain relatives or friends who

had enjoyed association with them likewise developed mediumistic talents.

Within two or three years a veritable spirit epidemic had spread through New York and adjoining states, east, west and south. Séances were the order of the day, or rather of the night; and all the technic and lingo which one may find in any present day textbook of spiritism were fully developed in that middle nineteenth century outbreak. Bereaved parents held converse with their spirit children in dialogues almost identical with those reported in "Raymond" between Sir Oliver Lodge and his departed son; ponderous objects acquired automotive qualities; under spirit influence the force of gravity was set at naught, or intensified a hundred fold, just as Mr. Crawford finds today in Ireland; currents of air, breezes from the beyond, fanned the faces of the faithful; impressive materializations took place; indeed the "direct voice" of the spirit was occasionally heard; nor were there wanting tokens of affection which under cover of darkness were discreetly bestowed on expectant sitters just as in the modern séance room.

The departed great "came through" in high-sounding platitudes, and the foundation principles of nature afterward were enunciated in language singularly out of keeping with the earthly history of the alleged communicants. In the old days, as today, the spirits of mischief were conspicuous and active, and there is ample documentary evidence set forth in the early fifties of the mad pranks of the *Poltergeist*, not only making night hideous but also involving the general dislocation of households and the destruction of property.

Let it be added that in those pre-Society-for-Psychical-Research days the religious factor soon came to be the driving force in the circles of the devotees; death had lost its sting, the grave its victory; the substance of things hoped for had gloriously become the sure evidence of things seen, heard and even handled; the very words which Sir Arthur Conan Doyle has used for a title to a recent book, "The New Revelation," were employed to denote the "new" gospel of 1850, which then just as now—although in the interim the fact seems to have been unaccountably forgotten—was destined to tower above and supersede all other forms of religion.

But we must not delay longer over this story of seventy years ago. It is only necessary to say that all minds were not equally credulous. Even in the early stages of the rappings in the Fox family, there were, as a contemporary critic remarks, ill-disposed and unbelieving people who faintly suggested the daughters as the cause, in spite of their protest of innocence. Eventually their exposure was complete. One investigator concludes his report, which reminds one of the inquiries of the Seibert Commission carried on a few years ago under the auspices of the University of Pennsylvania, with these words:

In conclusion, let me say, that according to my judgment, nothing of praeternatural or supernatural character took place; and that I was very strongly impressed with the conviction that the three women (Mrs. Fox and her two daughters) were shallow and simple cheats and tricksters, who, perhaps from an accident, had been led on by credulous people to impose upon them.

The Fox episode and its ending are worth bearing in mind because of the fact that notwithstanding the cheapness of their methods and the facility with which they were uncovered, one of the great exponents of the cult in our own day, no less a man than Conan Doyle, harks back to the Fox family as the legitimate sponsors of the new religion which is to regenerate the world. Sir Arthur uses these somewhat astonishing words:

The lowly manifestations of Hydesville have ripened into results which have engaged the finest group of intellects in this country during the last twenty years; and they are destined, in my opinion, to bring about by far the greatest development of human experience which the world has even seen.

ASPECTS OF SPIRITISM

The modern spiritistic period has been marked by three great waves: the inaugural midcentury wave I have just been discussing, the present postbellum flux, and midway between these the wave which followed the organization of the Society for Psychical Research.

In practice the traffic in spirits has taken on various aspects during this period. It has served as a mode of entertainment. Spiritistic performances have been put on for popular amusement as favorite parlor games. Heavy tables have acquired terpsichorean agility; blind-folded people have been constrained through "telepathy" to execute the will of the sitters; the ouija board has been brought into requisition as a faith-tinged pastime or sentimental diversion. On a larger scale, traveling showmen, with all the paraphernalia of the séance room, have staged exhibitions to delight the million. Such performers have outdone the Witch of Endor in their materializing accomplishments. The most remarkable of these shows in its time, perhaps for all time, was that of the clever handcuff kings, the Davenport brothers, who finally came to grief in their exhibition at Ithaca, N. Y., where some Cornell students made their way unobserved in the darkened room to the edge of the stage, and by turning on several strong flash-lights revealed the *modus operandi* of the mystical brothers to the satisfaction of all save those perchance who hunger and thirst after deception.

Second, the traffic in spirits has become an organized commercial undertaking. In addition to the itinerants whose business, like that of other conjurers, is to furnish a good show at a stipulated entrance fee, there are multitudes of settled mediums located in practically all the larger towns and cities who make their irregular living out of human credulity and gullibility. It is said that these imposters have a more or

less complete nation-wide organization, with central clearing houses for "spirit information" and elaborate lists of dupes in fact or prospect. This spirit trust has been discussed with considerable documentation by Edward H. Smith in the *Saturday Evening Post* of April 24, 1920.

Third, spiritism has become a country cousin of science. This phase of the movement stands to the "credit" of England where, in 1882, was formed the Society for Psychical Research. A similar organization sprang up in the United States in 1884. In this society were enrolled men prominent in science, literature and the professions. Records of unusual manifestations of all sorts were brought together; spiritistic experiments were conducted under "test" conditions which the experimenters considered satisfactory from the scientific point of view. The sincerity of the leaders of this movement is not to be questioned; and an enormous mass of curious literature represents the fruit of their labor and researches.

Finally, spiritism has become a new religion. This is the logical culmination of the spiritistic movement, as in fact the religious feature has always been the essential one whether recognized or not. Spirit circles have generally adopted the commoner religious rites and ceremonies; and as an intruder in the field of religion spiritism has ever aroused the antagonism of the churches, jealous of their own authority and methods. But now notwithstanding, in the aftermath of the world war, the formal attempt is being made to organize this great body of new-old doctrine into a new religious system. In the opinion of Conan Doyle: "No other religious movement in the world could put forward anything to compare with it." In his "New Revelation" this author vouchsafes to inquiring humanity a description of the life beyond which rivals in details that of St. John, the Divine; but it differs considerably from the latter, and, it may be added, is a trifle more in conformity with what may be taken to be present-day human aspirations.

CONTEMPORARY SPIRITISM AS A RELIGIOUS MOVEMENT

In dealing with the subject of contemporary spiritism, therefore, we must consider it as essentially a religious movement. As a religious development spiritism has followed traditional ways. In the history of religion magic was ever the starting point. In establishing communication between the devotee and the Deity magic rites, presided over by the head of the tribe, were the means resorted to, and were believed to have the virtue of controlling the operations of nature and compelling the favor of the Deity. As religious systems grow and mature along with the cultural progress of the race, magic rites tend to recede into the background, although they have never disappeared completely.

Numerous familiar forms and ceremonies in the church today are plainly reminiscent of the incantations of a cruder age.

The ordinary professional spiritistic séance is a very good replica of the magic rites of primitive society. Indeed the whole spiritistic movement is flagrantly atavistic and reflects the animism of the childhood of the race. Tylor remarked that the "spiritualistic theory belongs to the philosophy of the savages."

We have, then, as a starting point the two-fold fact: Spiritism is a religion; and it is a reflection of the religion of the most primitive stages of human society. This circumstance, that the subject is a religious one, hampers discussion somewhat, and has a peculiar effect on the attitude of the individual. There are certain subjects concerning which the majority of people have a curious mental bias which makes them more or less inaccessible to foreign and conflicting opinions. Among such subjects religion stands conspicuously foremost. Persons of diverging views on religion can rarely be brought to agree. The same may be true of politics, although to a lesser degree; while in the field of science this personal bent or bias is supposed to manifest itself least of all, the standpoint of the individual being determined by objective facts.

This peculiarity and the difficulty attached to the discussion of religious questions may be explained on the basis of the following considerations:

Religion in its highest reaches is essentially an expression or an outgrowth of the affective state of the individual. It is based on feeling rather than judgment, and the function of religion is to serve the most intimate personal needs of the devotee. In his classical treatise on the "Varieties of Religious Experience," Professor James uses these words: "The pivot round which the religious life, as we have traced it, revolves, is the interest of the individual in his private personal destiny. Religion in short is a monumental chapter in the history of human egotism."

Because of these two features, the dominant affective element and its identification with the vital self-interest of the individual, his religious belief may be said to be more a part of himself than any other item in his mental equipment. That it should be beyond the influence of attack or criticism follows naturally enough. These considerations are pertinent in the discussion of spiritism as in any other form of religion.

But just as the majority of people are not absorbingly religious, so the number of spiritists, although now declared to be increasing remarkably, is still relatively small in the total population. The question — What determines a man's deviation toward spiritism? — is only another form of the question — Why is one man more religious than

another? The matter of temperament here demands consideration. It is not too much to say that a person's temperament determines in general what his religious attitude will be. This fact was clearly set forth in Sir William Osler's splendid essay "Science and Immortality." You recall that he divided mankind into three classes: (1) the small band of devoted Teresians living strictly the religious life, willing to die for their belief if need be; (2) at the opposite pole the skeptical or disbelieving Gallionians; (3) while intermediate between the two and making up the bulk of humanity are the indifferent or neutral Laodiceans, who may lean perhaps one way or the other, according to circumstances, but who never go to the extreme of blind faith or agnostic scoffing, and remain more or less undisturbed and unaffected by the zealous activities of their neighbors on either side.

It is well to recognize, therefore, that personal attitudes typified by these three classes are not arrived at by processes of deliberation, logic and judgment, but are first and last questions of temperament, to change which lies not within the power of the individual. While the Laodicean may conceivably veer toward the point of view of the Gallionian or the Teresian, that either of the latter should be transformed would be almost miraculous, if not pathologic. "Let him that is scornful be scornful still" were words which had a deeper meaning than is usually supposed.

BELIEF IN SPIRITS BASED ON TEMPERAMENT

The theory of temperaments until lately had fallen somewhat into neglect in scientific circles, but is again coming into its own. Personality and character studies have led to the contemporary teaching of the *individual constitution* which is a definite and unchanging affair. It represents the hereditary and developmental neuropsychic attitudes, tendencies, and inclinations — the mental sets of the individual — which determine the way of his whole life and which he cannot escape.

This tyranny of our constitutional bents or mental tropisms must ever be borne in mind in considering a subject like the present one; and it must be further remembered that the constitutional trends with which we start early in life become fixed and reinforced as we go along, so that in our middle or later years we may be no more able to think or act out of harmony with them than to break through the steel bars of a prison.

Osler's categories apply equally to spiritism. Looking at the subject in this way it seems fair to conclude that the belief in spirits is not in the last analysis, as believers often take pains to tell us, an acquired belief quite foreign to their former mental habits and based solely on "evidence." Rather I think the belief in spirits is an act in faith temperamentally determined.

The biographies of some of the most noted modern spiritists throw light on this view. It is a matter of fact that all the best known exponents of this system of occultism have practically devoted their lives to the subject. Meyers, Hodgson, Barrett, Lodge, Doyle, all have spent many years in the spirit land. It is well to insist on this point, the significance of which is perhaps not sufficiently appreciated. Assuming the natural constitutional bent, these men having dedicated themselves to the inquiry, grew old in their quest, which assuming for them more and more importance as the years passed, eventually became a veritable obsession.

Quite naturally the leaders in spiritism look on the steps in their career simply as the gradual piling up of evidence which in the end forces conviction. They all emphasize this view of the matter. They take pains to refer to their earlier skeptical attitude, and to point out that they have not arrived at their conclusion prematurely. Being at length compelled to yield to the, to them, overwhelming mass of "evidence," they are at a loss to understand how any unbeliever reading their reports of this "evidence" can escape sharing their belief. But a consideration of the psychology of conviction is calculated to set the matter in a somewhat different light. Concerning his beginnings, Sir William Barrett in "The Threshold of the Unseen" says, "upwards of forty years ago I began the investigation of alleged supernormal phenomena with a perfectly detached and open mind." Referring to Dr. Hodgson, Sir Oliver Lodge remarks, "He devoted years of his life to the subject and made it practically his whole occupation." Conan Doyle in his "New Revelation" declares, "The subject of psychical research is one upon which I have thought more and about which I have been slower to form my opinion, than upon any other subject whatever." To some persons this may be a surprise. Those of us who have known Sir Arthur only as a physician and as the creator of remarkable characters in fiction are perhaps shocked to find that his type of mind should lend itself so readily to a spirit cult.

Hyslop speaks of the "conversion" of Doyle as a recent event which is calculated to help on the cause wonderfully. This view of Conan Doyle's position is obviously erroneous. In the book just referred to he shows that for more than thirty years he has devoted most of his spare time to psychical research; although "it is only within the last year or two that I have finally declared myself to be satisfied with the evidence." According to his own statement, therefore, the subject of spirit manifestation has been in the other's mind more than "any other subject whatever" for upward of a generation.

Conan Doyle indicates some of the steps by which he has arrived at his present position, and it is interesting to follow these. He finished

his medical education in 1882, "like many young medical men a convinced materialist as regards our personal destiny." He declares, however, that he was always "an earnest theist" (personal bent). At first he regarded the phenomena of spiritism as "the greatest nonsense upon earth." But he was thrown with friends who were seriously interested in the matter and whose integrity he could not doubt. His inclination also led him to read all the available literature on the subject. He was "amazed to find what a number of great men thoroughly believed that spirit was independent of matter and could survive it." Even at this early time when he was beginning his medical practice he was much more impressed by the attitude of Crookes, Wallace and Flammarion, who believed, than by Darwin, Huxley, Tindall and Herbert Spencer, who disbelieved. In this early choice what else can we see than an expression of the inherent tendencies of the individual mind which, adequately considered, might have foreshadowed to the psychologic observer the denouement thirty years later which Hyslop speaks of as the "conversion" of Conan Doyle?

The author attended some spirit séances at which tables were juggled about by unseen forces. Being convinced of the sincerity of the participants and unable to explain the phenomena, he declared himself "puzzled and worried." About this time he read "with interest and absolute skepticism" a book called the "Reminiscences of Judge Edmunds" wherein the judge told of his communications through many years, with his dead wife. Of his early séances Sir Arthur preserved notes. "I was still skeptical, but at least I was an inquirer." Moreover, the time that was not actually used for experiments was devoted to reading psychic literature. This reading, he says, "was continuous." The author had now arrived a little further along his predestined way. He had taken part in the reception of numerous spirit messages, some of which were trivial and irrelevant, while others appeared to carry the stamp of genuineness. He states that while he had no proof of their authenticity "they simply left me bewildered." The cumulative effect of the testimony of others began to make itself felt. The agreement of witnesses he felt constituted "some argument for their truth." He came to "appreciate more and more what a cloud of witnesses existed." It is needless to remark that the author's perspective was possibly not calculated to give him a just idea of the dimensions of this "cloud." As Professor Jastrow has remarked: "The obvious fact is strangely ignored that, for one exceptional scientist who subscribes to the reality of such communication, there are hundreds of equal authority who would violently resent the implication that they might be tempted to draw conclusions as to the nature of the universe from the testimony of mediums trafficking upon human credulity."

About 1891 Doyle joined the Psychical Research Society and had the "advantage" of reading all their reports, certainly no inconsiderable accomplishment. It does not seem possible that an investigator would have the patience to labor through this vast accumulation of literature if he were entirely dispassionate and not rather striving, however faintly, toward conviction. This state of mind the author had now reached. The influence of the society, he states, "was one of the powers which now helped me to shape my thoughts."

Sir Arthur was helped on a good way toward conviction by reading Meyers' "Human Personality." The thing which impressed him was the testimony concerning telepathy which he found himself ready to admit as an established scientific fact, in spite of dissenting views of the majority of scientific men. The acceptance of telepathy was a red letter day in Doyle's ghost-land travels. "The ground was cut from under the feet of the materialist and my old position had been destroyed." Having arrived thus far, one does not need to wonder that the few remaining steps were easy, nay inevitable. Starting with telepathy at one end of the series of psychic manifestations, the author found an unbroken chain of phenomena culminating in "actual manifestations of the spirit independently of the body."

MENTAL METAMORPHOSIS OF THE SPIRITIST

Given the necessary temperamental set, any subject which is strongly tinged affectively is likely to get itself so anchored in the subcellars of the mind as to be pursued to the same obsessional length as the spirit quest. Familiar examples are common enough in these days, to mention only certain types of prohibition apostles and vice crusaders, professional reformers, uplifters and self-appointed moral censors of various kinds, ultra-freudologists and antivivisectionists, Christian scientists, feminist extremists, pacifists and red socialists. Such men do not pursue their idea for a time as a fad or diversion and then change it for some other interest; on the contrary, their obsessing idea tends to become a life engrossment, reinforced with the passing years, and from its final utter domination we are probably safe in saying they could not free themselves if they would.

The career of Conan Doyle, as he has himself outlined it, is fairly illustrative of such a gradual mental metamorphosis. Over against his declaration, already quoted, that he had given more thought to psychical research than to any other subject whatever throughout his life, let us set the remark of another eminent scientist to illustrate this question of temperament we are discussing, which determines personal inclinations and attitudes, and which, with respect to any given theory, may make it at the same time impossible for the one mind to accept

and for the other to reject. The remark was made by Huxley in a discussion with Wallace on the topic of spiritism: "It may all be true for anything I know to the contrary, but really I cannot get up any interest in the subject."

To establish the underlying factors of faith we must study the personality of the believers rather than the reasons they bring forward in defense of their belief. It would be profitable to make such comparative personality studies of a group of religious devotees and a group of chronic skeptics. It is possible that their differential psychology might be found to be largely a matter of endocrine glands, smooth muscle fibers and the autonomic nervous system; and their mutual antipathies rather deducible from physiologic than logical factors.

But there is another feature which has been touched on but to which more specific reference must be made. This is the factor of *habit*. Sir Arthur repeatedly refers to his continued experiments and his continued reading along the chosen lines. He explains that it was his "interest" which kept him at it. This is, of course, true. We think and ponder over the thing we are interested in and continue to do so for the same reason. However, this continuance establishes a mental process which becomes, as we say, habitual, and which we know tends to be self-perpetuating even were the interest factor to diminish. In the case before us, however, interest and the habit tendency obviously work together and strengthen each other. After some years of this sort of thing the course of the mental operations of the individual is irretrievably fixed, and in nine cases out of ten he will follow his natural bent thus established to the end of the chapter.

Let us consider still another factor in the psychologic metamorphosis of conviction, namely, the striving, if one may so express it, of every thought process to arrive at a definite *goal*. Suspended judgment is a painful state; doubt and uncertainty are unpleasant at best and may become intolerable. It seems likely that in certain forms of mental disease definite relief is experienced when anxious uncertainty has been replaced in the patient's mind by the delusional interpretation on the basis of which he can again, after a fashion, adapt himself to his surroundings.

In the nature of things, religious and kindred doubts are the most painful of all. As far as the phenomena of spiritism are concerned, experience shows that inquirers after suspending judgment for a time during which they retain more or less the capacity for dispassionate critique — the skeptical stage, they usually call it — will generally one day end by accepting unreservedly the conclusions toward which their ideas have all along been tending.

The final stage in the mental transformation of the spiritist is that in which he reaches conviction, complete and unassailable, when he

passes from the stage of so-called scientific inquiry to that of absolute faith. This stage may be attained gradually and naturally without any special accelerating event. The affective coloring of the religious motive with its deep personal concern may be sufficient to usher the individual through the various stages until he has reached the final phase of spiritual calm, self satisfied in his clairvoyance of things past and to come.

For some, however, it may be that a special added motive is needed. The war has furnished this motive. For many the bereavement has been almost intolerable, the loss has seemed so unnatural, so cruel and useless. Both Sir Oliver Lodge and Conan Doyle lost a son in the war. In view of their previous history of spirit seeking, what more natural than that the pursuit should now be continued with redoubled zeal and devotion? That the power of self criticism under such circumstances may become very feeble, it is needless to say. The recent activities of both of these men, the inauguration of a new religion by Doyle and the publication of "Raymond" by Sir Oliver Lodge followed by his missionary trip to North America, are the logical sequels of their previous mental careers under the influence of the World War with its bereavements.

CRITERIA OF EVIDENCE ACCEPTED BY PSYCHIC WORKERS

That in the final religious phase of the gradual psychic metamorphosis we have been outlining the power of criticism is very weak indeed is evidenced by the fact that the exposure of fraud has little or no effect on the faith of the devotee. Thus, after the cheap trickery of the notorious Eusapia Palladino had been brought to light and she herself had been discredited, and although, as Hyslop admits, 300 members of the Society for Psychical Research deserted the spirit camp following these events, Sir Oliver could still say, "I am, therefore, in hopes that the present decadent state of the Neapolitan woman may be only temporary, and that hereafter some competent and thoroughly prepared witness may yet bring testimony to the continued existence of a genuine abnormal power in her organism." Can such a sentiment as this express anything else than a downright headlong will to believe in spite of everything?

Sir Oliver describes another incident which might have shaken the faith of any not unwilling to be undeceived. F. H. W. Meyers had written a message which he had delivered in a sealed envelope to Sir Oliver, to be used as a test after the writer's death. Shortly after this event the message was subjected to mediumistic influence, but not until fourteen years had elapsed was the time deemed ripe to make trial whether the contents of the sealed envelope had been telepathically discerned. The result was complete failure. A sealed message written

with similar intent by Richard Hodgson is in existence, and a reward of \$1,000 has been offered to any medium who can reveal it. The reward remains unclaimed.

The quality of the faculty of criticism when under the coercion of faith is further illustrated by the acceptance of the alleged messages from Meyers by Sir Oliver as genuine, although the former was unable during fourteen years following his death to infuse into the mind of any medium the contents of the sealed message which he had expressly prepared for that purpose, and in face of the testimony of Meyers' widow on the subject of these messages, in which she declared, "after a very careful study of all the messages we have found nothing which we can consider of the smallest evidential value."

Reference might here be made to the findings of that indefatigable promoter of things psychic, Mr. Hereward Carrington, in his compilation "The Physical Phenomena of Spiritualism." This authority essays to distinguish "fraudulent" from "genuine" phenomena. In a text of 417 pages he finds it necessary to devote 318 to the former. Those who regret that Mr. Carrington's "genuine" phenomena were only sufficient to fill one fourth of his book, are doomed to further disappointment when they come to this statement (p. 336): "There may be much fraud in modern spiritualism, in fact, I am disposed to believe that fully 98 per cent. of the phenomena, both mental and physical, are fraudulently produced, but," etc.

The disappointment of the reader seeking light in this volume will be grievously supplemented by bewilderment when he discovers that the difference between the "fraudulent" and "genuine" manifestations, for aught that is revealed in the text, lies secluded in the psychic eye of the author. The same kinds of "tests" are described in both sections of the book, the distinction being that the author believes in the one group and disbelieves the other. Moreover, in the "fraudulent" section there are pages devoted to the trick methods by which similar phenomena recorded in the "genuine" section can be produced.

Finally the seeker's disappointment and bewilderment will merge in confused despair when he realizes that the "genuine" matter is made up all but entirely of a series of quotations. Why should these devoted researchers be able to develop no manifestations of their very own? Hidden away in the mass of this book are two pages in which Mr. Carrington records the sum of his personal psychic experiences. These consisted in certain rappings heard in his room at night at about 10 or 11 o'clock and continued until he went to sleep. These noises, which he avers did not sound like creaks, recurred during four or five weeks; one night he located them on his mantel piece, and laying his hand thereon, felt distinctly the vibration of the wood. Therefore Mr.

Carrington believes in raps. He admits, however, that he could get no intelligence from them. Also, he has sat by the hour over the planchette, but without result. At length one night, at 11 o'clock, his eyes being too tired to permit him to work, he again resorted to the mystic apparatus. By 2 a. m. he had obtained "a few vague scratches." As he was about to give up in disgust there came on the planchette board a few faint raps; then again continued silence. Verily the progeny of the laboring mountains should not after all have been regarded so contemptuously.

Were it desirable to examine further the criteria of evidence which satisfy the psychic researcher, we might cite almost at random the spoken and written words of the author of "Raymond." In an earlier work, "The Survival of Man," Sir Oliver gives his qualifications as an authority on spirit manifestations. "A physicist," he declares, "can make no assertion on it one way or the other. . . . As a physicist I do not know; these are not processes I understand." But spirit information being of a different brand must needs come by other channels than those by which all the rest of our experiential data are acquired; and where faith enters in the censor, logic, nods. Sir Oliver accepts telepathy, but not by the methods of demonstration or the processes of reasoning by which he accepts the facts of science. Such rigid criteria he gives over entirely. He has experimented with telepathy but states that he has not been able personally to demonstrate it. As a "percipient" he failed. For example, when the "five of diamonds" was telepathed to him he got "scissors." As "agent" he likewise failed in the majority of cases. He has known of happenings, however, of which, to the neglect of the law of parsimony, telepathy has seemed to him the sole explanation; and for proof and reason he can only say: "to the best of my scientific belief no collusion or trickery was possible." He had expressed similar belief in Eusapia Palladino.

Let me add a single quotation from "The Survival of Man" (p. 321), which most strikingly exemplifies the polar divergence between the method of arriving at scientific conclusions and that of reaching spiritistic conviction.

The old series of sittings with Mrs. Piper convinced me of survival, for reasons which I should find it hard to formulate in any strict fashion, but that was their distinct effect. They also made me suspect—or more than suspect—that surviving intelligences were in some cases consciously communicating—yes, in some few cases, consciously; though more usually the messages came in all probability from an unconscious stratum, being received by the medium in an inspirational manner analogous to psychometry.

Would one suspect that the foregoing sentence had been written by a scientist?

A certain characteristic religious intolerance also begins to make its appearance in the writings of this author. In "Raymond" he refers to the "little systems" and "contemporary blindness" of those who do not subscribe to his hypothesis; he speaks of the "assumptions and blind guesses" of men of the materialistic school, and adds this further vocal boomerang, "their device being to anticipate and speak of what they hope for, as if it were already an accomplished fact."

Professor Jastrow, commenting on the type of mind revealed in these various quotations, says:

The phenomenon is a puzzling one; for we associate with the effect of a professional training a general robustness of logical vigor, a thorough saturation of the mind in all its vocations with the habits of rigid evidence and critical caution. We assume a consistency of mental habit, and in that assumption seemingly go astray. We must make room for the existence of minds streaked with rationality but not uniformly penetrated by the stabilizing quality; we must consider reserved areas of prejudice and predilection in which ideas flourish and convictions are cherished with slight regard to their reconciliation with the dominant logicity of the rest of one's beliefs.

But enough has been said to indicate quite clearly that in dealing with the spiritistic theory, we have to do in the main with matters of faith and religious aspiration rather than with the processes of dispassionate scientific inquiry. Indeed the religious climax of the spirit quest following the world war is clearly expressed by Sir Oliver. "For many years I held my tongue but I have come out since the war more into the open because of the extensive bereavement which could be comforted." Here we see the ultimate end of all religious systems, to bring spiritual consolation and assurance which shall satisfy the personal longing and self seeking out of which all religious systems develop.

Conan Doyle similarly describes the effect of the war in his own case. Had it not been for this cataclysm, he says, he might have "drifted on for my whole life simply as a psychic researcher." But in the midst of the agony of war bereavement, "I seemed suddenly to see that this subject with which I had so long dallied was not merely a study of a force outside the realms of science, but that it was really something tremendous, a breaking-down of the walls between two worlds, a direct undeniable message from beyond, a call of hope and of guidance to the human race at the time of its deepest affliction." This language might fit beautifully into an impassioned religious exhortation but it has nothing to do with the calm consideration of scientific facts. The author is no longer an inquirer but a believer. The labor of investigation is over. The voyage is done and the believer is safe in the harbor of his spiritual revelations. As the author expresses it, "the objective side of it ceased to interest, for having made up one's mind that it was true, there was an end of the matter. The religious side of it was clearly of infinitely greater importance."

The *age* factor has only indirectly been referred to. This must also be taken into account. No explanation of the fact that religious reflections and practices tend to increase with advancing years is necessary; not only do latent tendencies grow active, but there may take place what at first glance appears to be a constitutional transformation, in that a strikingly irreligious youth is succeeded by a devout old age, bitterly repentant of the "errors" of earlier life. These epochal changes are common enough and will be found not to controvert the idea of temperaments and mental sets which we have been developing. These circumstances may not be of major importance in the present question, but what is of importance is the fact that habitual mental reactions tend to become unalterably fixed in later life, due not only to the habit factor itself in its anatomic and physiologic bearing, but *pari passu* to the loss of psychic resiliency in old age with its intolerance of the unaccustomed. It is doubtful if the old man, or any of us who are getting on, appreciates what a slave he is becoming to his habitual tendencies in thought and conduct. For the person who has grown old in a religious belief, spiritistic or otherwise, true or false, there is probably no mutation possible.

SUMMARY

We have come to the final step of our inquiry. We have seen that the leaders of the spiritist movement are men of a certain disposition of mind which predisposes them to this pursuit, which they follow with devoted and increasing interest throughout their lives. The motive of this interest is religious craving, the seeds of which are sown in every breast. For some the age-worn forms of orthodox religion are unsatisfying, and a pseudo-scientific garnish like that of psychical research supplies the necessary appeal. The mental metamorphosis of the spiritist is not difficult to trace. He feels himself at first a skeptic, then a curious onlooker, then an interested inquirer, then an earnest seeker; and finally by this declension a devout believer. If he is called on to describe the process he declares that he has approached the subject with an open mind, and that steadily accumulating "evidence" has at length inevitably forced conviction on him without any motion of his own. What he will not tell you is that having a personal bent for the occult or the shady side of science, the demands of his nature have forced him to devote time and attention in increasing measure to these matters; that the driving force of ultimate personal need has caused him to discern evidence where another might have found only accident, coincidence, or utter irrelevancy; and that under the enslaving influence of habit crystallized by the passing years, what began as a more or less dispassionate inquiry has ended as a veritable spiritual quest, pressing onward even to the threshold of the abnormal.

Are the celebrated men who lead the spiritist movement justified in their public attitude and propaganda? Their own answer to this question has been heard. They hold themselves to be the ministers of a new religion more important than any the world has ever seen, a religion not only destined to supersede all others and bring final peace and satisfaction to humanity while in the flesh, but which answers better than any other the ingrained human aversion to extinction, and which vouchsafes a livelier picture than any other religion of the assumed existence beyond the grave. With such a conception of their high mission, these men cannot do otherwise than go out into the world and preach their gospel. Such an act is merely reflex to the circumstances which have led up to it.

There are certain consequences, however, of which we are bound to take account. Sir Arthur and Sir Oliver may be honest and devout, but the school of spiritism is broad and shelters many less luminous characters. The traffic of the ghost monger is notorious and baneful, and the encouragement which these shady characters receive from the campaign of the leaders of the psychical movement cannot but be lamented.

The point has been raised that this new religious system gives comfort to the bereaved at the time of greatest need. The leaders expressly bring forward this as the supreme object of their work. How far this attitude is justified may be open to serious question. There is little doubt that the bereaved parent concentrating emotionally on the memory of the dead child may eventually come into a mental state in which he believes himself in relationship with that child or in which he may even catch a vision or hear his voice. These abnormal states are familiar in mental medicine, and while by cultivating them a certain amount of satisfaction may be gained, it is legitimate to ask whether this is the wholesome and most suitable method of dealing with states of bereavement and sorrow. It may be that certain otherwise stable minds may indulge in these operations, derive a degree of comfort and suffer no serious injury; for others, however, the procedure is distinctly unwholesome, if not dangerous, and to encourage it is to take risks unwarranted by any established facts.

BLOOD SUGAR STUDIES IN DEMENTIA PRAECOX AND MANIC-DEPRESSIVE INSANITY*

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A study of a number of cases of dementia praecox and manic-depressive insanity from the point of view of blood sugar tolerance, as determined by means of Benedict's¹ modification of the Lewis-Benedict method for the determination of sugar in the blood, has been made. Such a study was regarded as of special interest because of the apparent meagerness of mention in the literature of previous work in this field. At the onset of our experiment the only reference of any pertinence was that of Weston² in which presentation is made merely of single (apparently nonfasting) blood sugar determinations upon a series of patients classified essentially upon the basis of ward behavior, rather than of actual clinical status. In another article appearing since, Weston³ reports a second series of cases of dementia praecox and manic-depressive insanity in which single fasting bloods were examined for glucose according to the technic of Meyers and Bailey, and in which apparently no essential deviation from normal was determined. We agree that the fasting levels are well within the normal range and that they in themselves carry little or no diagnostic value. On the other hand, we do believe that a series of determinations which shows how the body handles glucose in a comparative way over a given period of time may be of diagnostic value, at any rate of great physiologic importance. We may mention that Weston in his first paper also used the Meyers and Bailey⁴ blood sugar technic which is a modification of the Lewis-Benedict method. It is essentially the same as the Benedict modification of the Lewis-Benedict method except that it provides for less dilution of the blood, so that the final reaction may take place in a more concentrated mixture of glucose and picric acid and yet avoids the long process of boiling to dryness as indicated in the original method. Benedict uses a dilution of 1 to 12.5 while Meyers and Bailey use a dilution of 1 to 5.0.

* From the State Psychopathic Hospital, Ann Arbor, Mich.

1. Benedict, S. R.: A Modification of the Lewis-Benedict Method for Determination of Sugar in the Blood, *J. Biol. Chem.* **34**:203 (April) 1918.

2. Weston, P. G.: Sugar Content of the Blood and Spinal Fluid of Insane Subjects, *J. M. Research* **35**:198 (Nov.) 1916.

3. Weston, P. G.: Analyses of Blood of Insane Patient. *Arch. Neurol. & Psychiat.* **3**:147 (Feb.) 1920.

4. Meyers, V. C., and Bailey, C. V.: *J. Biol. Chem.* **24**:147, 1916.

An extensive communication by Kooy,⁵ appearing as the present paper was being prepared for publication, reports a series of cases of dementia praecox, epilepsy, dementia paralytica, "anxious melancholia" and "nonanxious melancholia," in which carbohydrate tolerance tests were performed. Fasting bloods were taken; the patient was given breakfast consisting of 100 gm. of bread and 200 c.c. of milk; bloods were then drawn every three-fourths hour for two and one-fourth hours and examined according to the Bang technic. The author attempts to show that the various groups named above give characteristic curves. A glance at the composite curves illustrated by Kooy on page 281, reveals that all curves are practically parallel and that the only differential point is the acme level. The author asserts that this difference in acme level is due to the different emotional character of the diseases. He then gives a detailed discussion on epinephrin in different emotional states and its importance in mobilizing the glycogen of the liver in consequence of which more glucose is found in the peripheral blood. He further discusses the "piqûre" of Claude Bernard and the "piqûre" of Sachs-Aronsohn causing hyperglycemia, showing that the latter is effected by a hypersecretion of epinephrin. Nothing is said, however, of a prick of the skin causing a varying output of epinephrin depending on the emotional reaction of the patient. We shall later discuss the lack of differential value of the curves he obtained. Further, although this metabolism experiment is well controlled from the standpoint of disease, temperature, environment, rest of the patient during the experiment and the patients' age, there is absolutely no control as to the subject's weight. At least no account of it is given in the paper. This certainly is a serious criticism when we know how important the surface area is in all metabolism experiments.

PROCEDURE

The exact procedure in our work was, for the sake of uniformity, essentially that recommended by Janney and Isaacson⁶ in April, 1918, which seems to have become practically standard for this type of investigation. Thus each patient was required to fast from 7 p. m. of the evening preceding the test day until the completion of the test on the succeeding morning. At 7 a. m. on the test morning, vein puncture was performed and the patient was required to ingest a definite weight of glucose (1.75 gm. per kilogram of body weight) dissolved in 2.5 c.c. of distilled water per gram of glucose. Blood was then drawn for three successive hours following the administration of the glucose. The urines were tested for glycosuria in all cases. The actual technic

5. Kooy, F. H.: Hyperglycaemia in Mental Disorders, *Brain* **42**:214, 1919.

6. Janney, N. W., and Isaacson, V. I.: A Blood Sugar Tolerance Test, *J. A. M. A.* **70**:1131 (April 20) 1918.

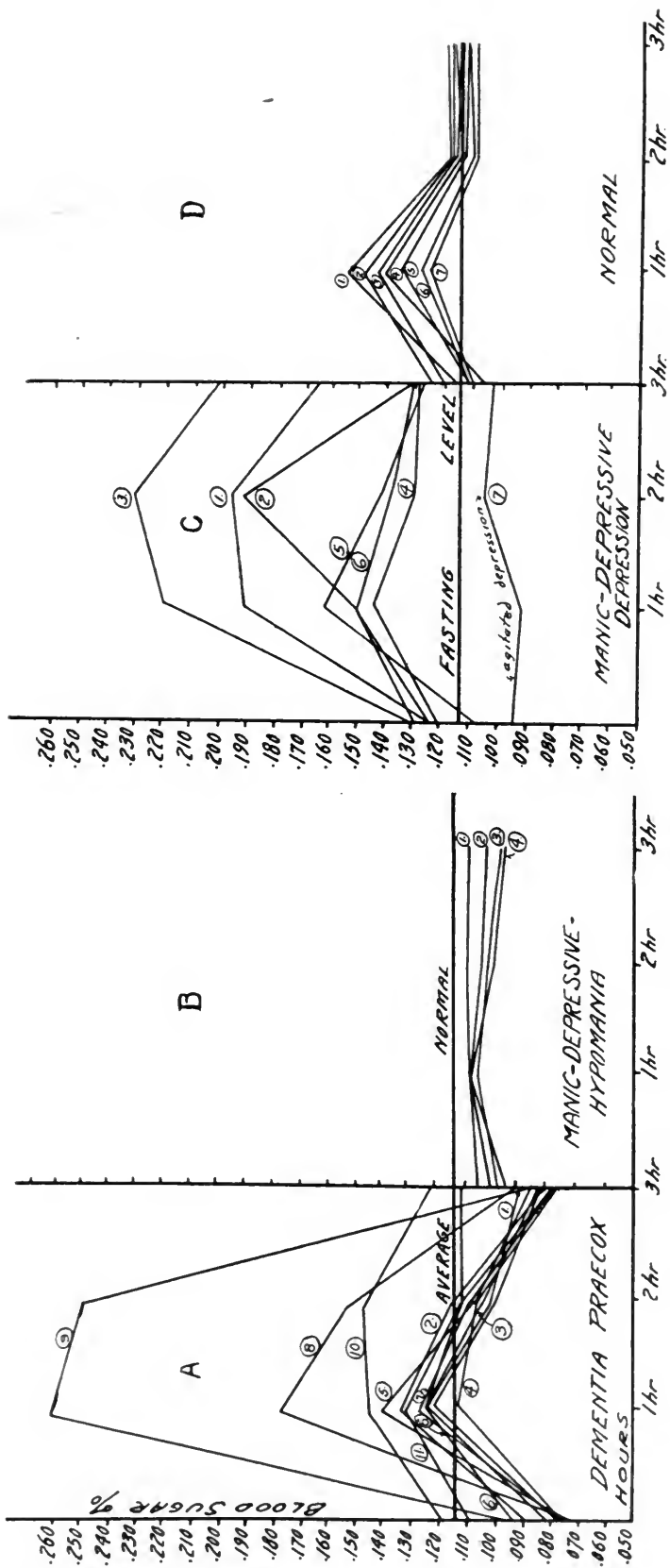


Fig. 1.—Composite sugar tolerance curves for normal and psychotic subjects.

employed for the quantitative determination of the blood sugar was essentially that of Lewis-Benedict, which was specifically as follows:

Two c.c. of blood are drawn into an Ostwald pipet, containing a little powdered potassium oxalate, and discharged into a 25 c.c. graduated flask. The pipet is twice rinsed out with distilled water, these washings being added to the blood. After a minute or two the blood is practically completely laked. A solution of sodium picrate and picric acid is added to the 25 c.c. mark (using a few drops of alcohol to dispel the foam if necessary) and the mixture thoroughly shaken. (We never used any form of alcohol or ether for anti-foam, because our experimental work soon showed that these substances gave the same color reactions as glucose.) After a minute or two (or longer) the mixture is poured on a dry filter and the clear filtrate collected in a dry beaker. Exactly 8 c.c. of the filtrate are measured into a large test tube bearing graduations at 12.5 c.c. and 25 c.c., and 1 c.c. of 20 per cent. anhydrous sodium carbonate solution is added. The tube is plugged with cotton and immersed in boiling water for ten minutes. It is then removed and the contents are cooled under running water and diluted to 12.5 c.c. or 25 c.c., the amount of dilution depending on the depth of color. At any time within half an hour the color solution is compared in a colorimeter with a suitable standard solution, the standard being set at a height of 15 mm.

The solution of sodium picrate and picric acid is prepared thus: Place 36 gm. of dry powdered picric acid in a liter flask, add 500 c.c. of 1 per cent. sodium hydroxid solution and 400 c.c. of hot water. Shake occasionally until dissolved, cool and dilute to one liter.

The picric acid obtained since the war contains varying amounts of water; therefore, it cannot be accurately weighed. Benedict has determined that the above solution when properly made has an acidity of 0.04 N. with phenolphthalein used as an indicator. Therefore, all our sodium picrate solutions are made approximate and titrated against a standard alkali and corrected to 0.04 normal solution.

Our picramic standard was not made according to the formula given by Lewis-Benedict. We learned from experience that the quality of picramic acid varied with different firms—thus the color standard would vary. Therefore, our initial color standard was a known glucose (0.1 per cent.) made up from chemically pure glucose and run through the technic. Our permanent standard, namely picramic acid standard, was made approximate and then diluted till it checked colorimetrically with a known glucose standard previously established. Our picramic standard calculates about 70 mg. of picramic acid to 1 liter of distilled water.

BLOOD SUGAR DETERMINATIONS IN NONPSYCHOTIC PERSONS

There are several precautions to be taken in controlling the experiment:

1. The persons examined must not have any bodily diseases which are apt to influence the blood sugar record, such as brain tumor, fevers, nephritis, pancreatitis and great loss of blood.

2. The body temperature of individuals examined must be within normal range; all febrile cases are excluded.

3. The temperature of the environment must be practically constant for all cases examined. Therefore, all subjects were kept in the ward till the examination was complete.

4. No control case was used in which there was fatigue or exhaustion from overwork or lack of sleep. Those subjects whose resistance was apparently below normal were not used as a control. Previous

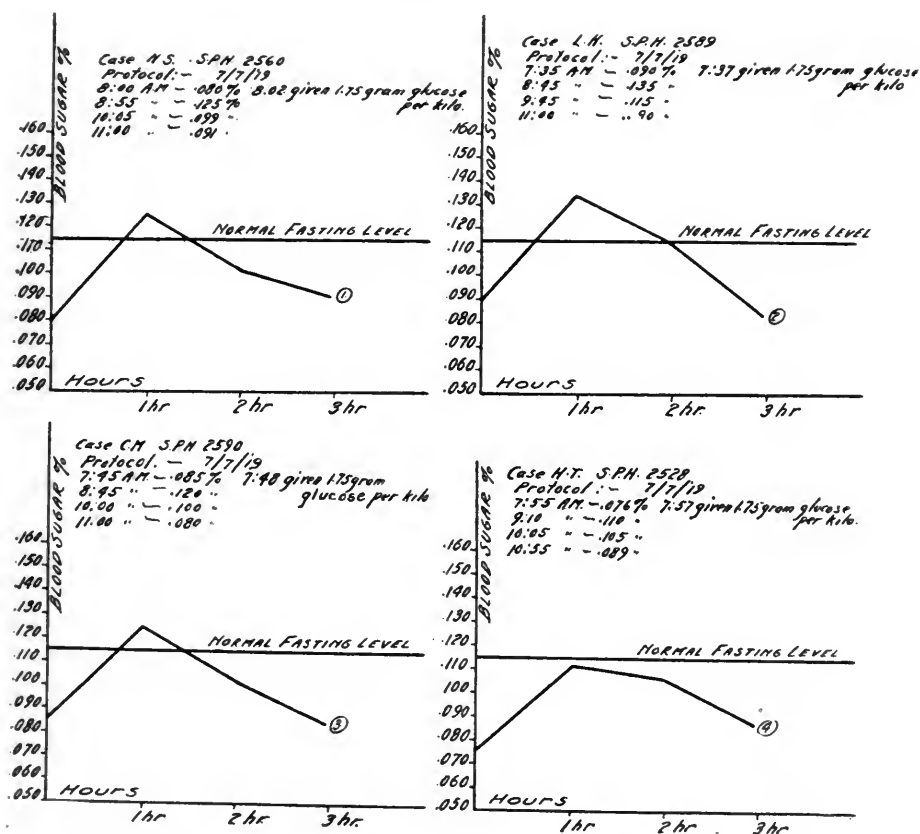


Fig. 2.—Individual sugar tolerance curves in dementia praecox.

work has shown that such cases show a lower fasting blood sugar record than normal. Fatigue, exhaustion and lack of sleep may all enter into the psychotic cases, often as a symptom of the disease.

5. Age we also know is of importance in blood sugar records. As persons grow old they frequently show a higher threshold and thus a higher blood sugar record. Our controls were chosen to include the same age range as that of the psychotic cases.

6. Of course, no control patient was permitted to work. The psychotic patients conducted themselves as usual.

7. None of our psychotic patients were obese and none were emaciated. Therefore, these two groups were not included in our controls. Weight was further controlled by giving the patient 1.75 gm. of glucose per kilogram of body weight, it being well known that even normal persons have a different rate of metabolism, which in turn bears some relation to weight.

8. Finally, vein puncture was performed by a person especially skilled in that technic and all patients were punctured by the same person, thus reducing as much as possible the emotional disturbance that might be incident to the experiment. This phase of the experiment cannot be controlled as well as one would wish.

The curves obtained in this series are given in the composite chart, (Fig. 1D). The numbers indicating the various curves correspond to the respective cases.

PROTOCOLS

CASE 1.—M. C., man, aged 55. 7 a. m.: Control blood, 0.115 per cent. 7:02 a. m.: Given 1.75 gm. glucose per kilogram. 8 a. m., 0.152 per cent.; 9 a. m., 0.120 per cent.; 10 a. m., 0.118 per cent.

CASE 2.—J. D., man, aged 35. 6 a. m.: Control blood, 0.125 per cent. 6:02 a. m.: Given 1.75 gm. glucose per kilogram. 7 a. m., 0.147 per cent.; 8 a. m., 0.117 per cent.; 9 a. m., 0.116 per cent.

CASE 3.—M. F., woman, aged 20. 7 a. m.: Control blood, 0.121 per cent. 7:02 a. m.: Given 1.75 gm. glucose per kilogram. 8 a. m., 0.140 per cent.; 9 a. m., 0.115 per cent.; 10 a. m., 0.116 per cent.

CASE 4.—H. A., woman, aged 19. 7 a. m.: Control blood, 0.105 per cent. 7:03 a. m.: Given 1.75 gm. glucose per kilogram. 8 a. m., 0.135 per cent.; 9 a. m., 0.113 per cent.; 10 a. m., 0.111 per cent.

CASE 5.—C. H., man, aged 30. 7 a. m.: Control blood, 0.112 per cent. 7:03 a. m.: Given 1.75 gm. glucose per kilogram. 8 a. m., 0.131 per cent.; 10 a. m., 0.108 per cent.; 11 a. m., 0.110 per cent.

CASE 6.—S. H., man, aged 19. 8 a. m.: Control blood, 0.112 per cent. 8:03 a. m.: Given 1.75 gm. glucose per kilogram. 9 a. m., 0.123 per cent.; 10 a. m., 0.108 per cent.; 11 a. m., 0.110 per cent.

CASE 7.—D. T., man, aged 25. 8 a. m.: Control blood, 0.110 per cent. 8:03 a. m.: Given 1.75 gm. glucose per kilogram. 9 a. m., 0.122 per cent.; 10 a. m., 0.107 per cent.; 11 a. m., 0.107 per cent.

PSYCHOTIC GROUP

This group includes eleven cases of dementia praecox and eleven cases belonging to the group of manic depressive insanity. Of the latter group, four patients were of the hypomanic phase and five were typically of the depressed phase. Two other patients, Cases 3 and 7, presented a somewhat atypical form of depression, but clinically seemed to belong in this group.

The exact psychiatric status of the cases may be determined from the accompanying abstracts of case reports, all of which were kindly prepared by Dr. A. M. Barrett.

The individual protocols and curves are represented graphically by Figures 2 to 7, and in the composite chart (Fig. 1).

DEMENTIA PRAECOX GROUP

Eleven cases are included in this series. Nine patients (Cases 1 to 9) were in acute phases of the disease and two (Cases 10 and 11)

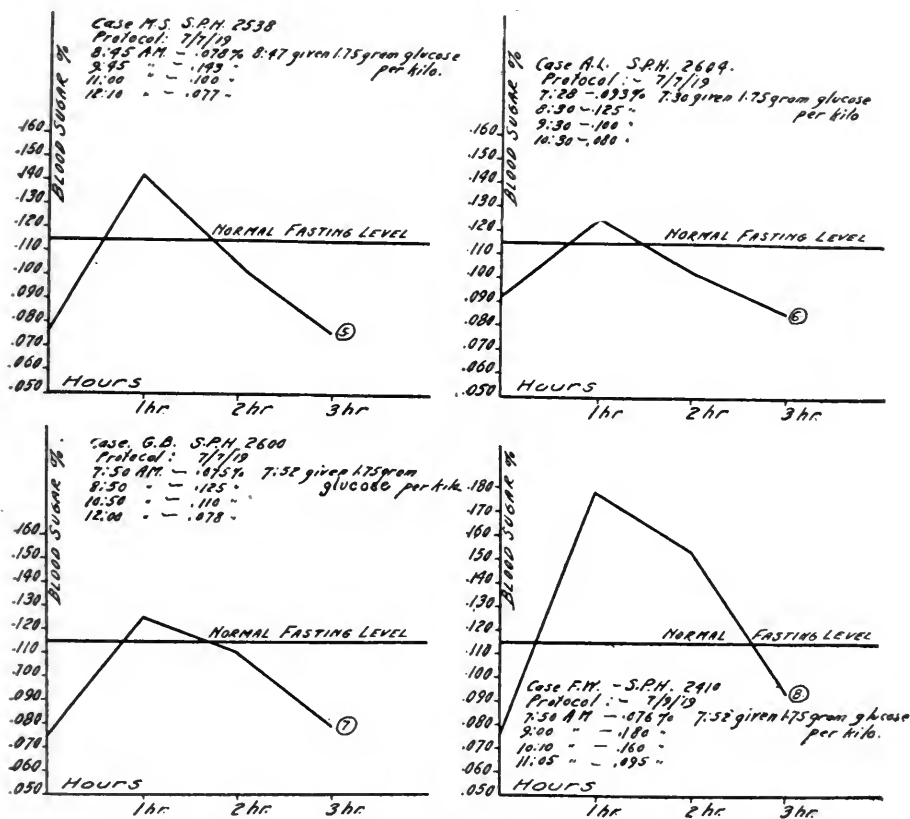


Fig. 3.—Individual sugar tolerance curves in dementia praecox.

had for some time shown no acute symptoms and were in a condition of mild deterioration (Fig. 1A, Curves 1 to 11; Figs. 2, 3 and 4, Curves 1 to 11).

CASE 8 (S. P. H. 2560).—Diagnosis: Dementia praecox, paranoid type (Fig. 2, Curve 1). N. S., a man, aged 25, admitted to the State Psychopathic Hospital, Ann Arbor, April 29, 1919, whose family were free from nervous or mental disorders, had always been considered an odd individual. He never mixed well with other people and kept himself isolated in the family.

He was drafted into the National Army and sent to Camp Custer on Aug. 28, 1918. He showed considerable anxiety over this, and remarked that he wished that he had married, as that would have kept him out of danger. About that time a relative had been drowned from a torpedoed transport and he seemed to fear that the same fate awaited him. At Camp Custer he did not do well and on Jan. 8, 1919, he was discharged and returned to his home. There he was apathetic, showed no inclination to find work and spent most of his time in his room, smoking incessantly. On his admission to this hospital he was irritable, resented having been brought here, and refused to cooperate in the examinations. For the first few days, he was surly and threatening in his attitude. There was marked disinterestedness in his surroundings, his chief reaction being resentment toward his friends and a disinclination to talk of any personal matters. His spontaneous interests were centered in some financial dealings with his sister over a piece of property. Whenever this was discussed, his irritability was increased. This attitude continued for about three weeks, when he showed some oddities of behavior. He would sit for long periods apparently engrossed in thought. Occasionally he would go to his room and perform various calisthenic exercises.

On June 1, he talked somewhat more freely. He spoke of a fear he had while in the army that he was suspected of being a German spy, and that he would be shot. He recently became quite excited and threatened his brother for having beaten him out of money. At night he talked while seemingly asleep. What he said related to his resentment toward his family. At times he would burst out in boisterous, unmotivated laughter.

The physical examination revealed no important pathologic condition. The Wassermann reaction on the blood was negative. There were no neurologic abnormalities.

On July 22, 1919, he was transferred to the state hospital at Kalamazoo.

CASE 9 (S. P. H. 2589).—Diagnosis: Dementia praecox, hebephrenic type (Fig. 2, Curve 2).

L. H., a man, aged 19, was admitted May 30, 1919. Aside from the death of a paternal grandmother in convulsions during pregnancy, the family was free from mental or nervous disorders. During infancy he was extremely ill for a long period from gastro-intestinal disorders. He was a weak, anemic child and did not enter school until the age of 7. He continued as an average scholar until he reached the eighth grade at the age of 15. Temperamentally he was quiet, seclusive and was regarded as a "home boy." After leaving school he became a machinist, continuing at this until the age of 19. One week prior to admission, he complained of feeling tired, he did not eat any supper and that night he did not sleep. During the night he appeared to be delirious and imagined that he was being chased by someone who was trying to kill him. He complained that his persecutors had murdered others and were trying to put the responsibility onto him.

For several days he was apprehensive and much perplexed. One day he stripped off his clothing and left the house nude, saying God told him that he did not need clothing, that he would be given a suit of armor and a crown with a star.

On the fourth day after the onset of the mental disturbance, he became so resistive and antagonistic that it became necessary to place him in confinement. There he was noisy and destructive. On the fifth day he was brought to the hospital. Here he was apprehensive, perplexed and constantly hallucinated. His stream of thought was much interrupted by hallucinations

and blocking. He spoke of spirits bothering him. His mind was troubled all the time. He thought he was not on earth. He rapidly developed a marked emotional let-down and took no interest in anything about him. He stood about in attitudes as if abstracted and often could be observed laughing in a silly manner without provocation.

Aside from poor nutrition there were no serious physical or neurologic abnormalities.

CASE 10 (S. P. H. 2590).—Diagnosis: Dementia praecox, simple deterioration (Fig. 2, Curve 3).

C. M., a man, aged 22, was admitted June 3, 1919. The family history was negative. There seemed to have been no serious mental disturbance until

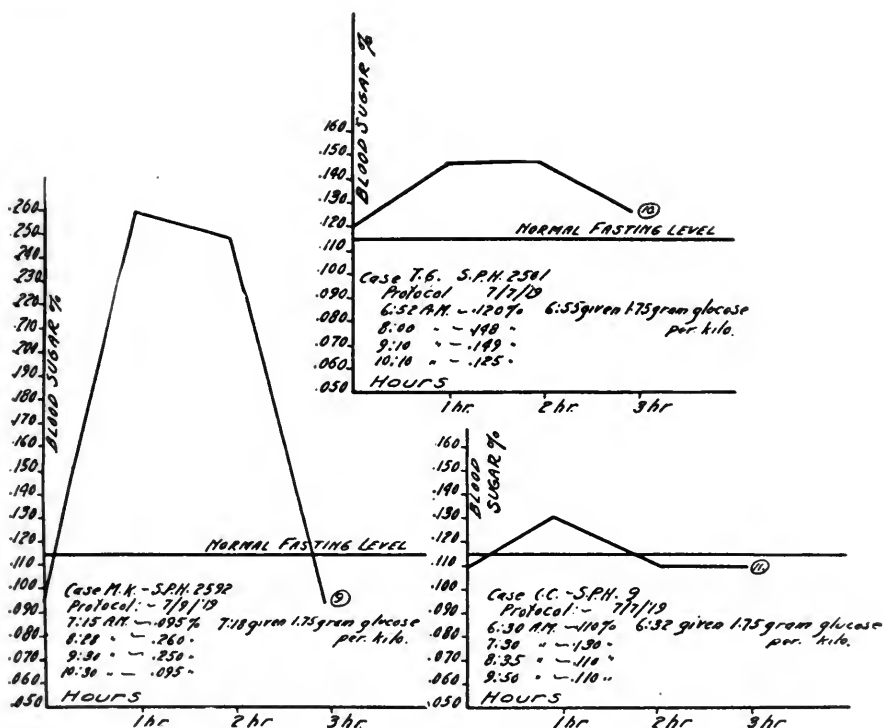


Fig. 4.—Individual sugar tolerance curves in dementia praecox.

his discharge from the army in February, 1919. Following this he became irritable and forgetful. He developed peculiar behavior which led to his commitment to this hospital. Here his attitude was one of marked indifference. He was seclusive, talked but little, and then his replies were superficial and inadequate. He sat about as if abstracted. At times he laughed without provocation. The only delusion that was expressed was in regard to his mother. He denied that she was his mother and became irritable if the subject was discussed. There were no hallucinations while he was under observation.

There were no serious physical or neurologic abnormalities. The Wassermann reaction on the blood was negative.

CASE 11 (S. P. H. 2528).—Diagnosis: Dementia praecox, hebephrenic type (Fig. 2, Curve 4).

H. T., a woman, aged 27, was admitted March 14, 1919. She had a bad home environment during childhood. Her parents separated, and her mother was immoral. At the age of 12, she was sent to a state industrial school where she remained until she was 18. After this she was employed at various occupations. At the age of 25 she became nervous and complained of various transitory pains. These continued until the age of 27, when she was sent to a general hospital because of a pain in the inguinal region.

A few days later she became acutely disturbed. She showed extreme perplexity, marked blocking of thought, ambivalence and auditory hallucinations. The content of thought was much taken up with erotic phantasies and self reproaches for these. She spoke of delusions of influences; a machine of some sort was drawing her head. This attitude continued for several months, when there occurred episodes of silly laughter and careless behavior. After a few weeks this passed away and she again became perplexed and hallucinated and showed marked erotic conduct. She is still in the hospital somewhat less perplexed, but there is still evident blocking and numerous schizophrenic symptoms.

The physical examination revealed no pathologic condition.

Neurologic examination revealed slightly irregular pupils, fine tremors of the tongue, and absence of abdominal and epigastric reflexes.

CASE 12 (S. P. H. 2538).—Diagnosis: Dementia praecox, catatonic type, mild stupor (Fig. 3, Curve 5).

M. S., a woman, aged 37, was admitted March 29, 1919. The family history was negative. She was married but had no children. Until eight months before admission she was free from physical or nervous illness. At that time she was in a slight automobile accident from which she soon made a good recovery. About Oct. 1, 1918, she expressed delusions of infidelity on the part of her husband, and complained of being influenced by others around her. She thought she had been hypnotized. This continued until her admission here. At that time she appeared as if in a daze and showed blocking. She became more inactive, spoke but little and then in hesitating fragments. She remained for brief periods in cataleptic attitudes. At times there were impulsive outbursts of excitement.

At the time of the tests she was unproductive, resistive and showed but little interest in her ward surroundings.

There were no physical or neurologic abnormalities. The Wassermann reaction of the blood was negative.

CASE 13 (S. P. H. 2604).—Diagnosis: Dementia praecox, hebephrenic type (Fig. 3, Curve 6).

A. L., a man, aged 27, was admitted June 22, 1919. The family history was negative. The patient had had a common school education. In September, 1917, he entered an army training camp. In March, 1918, he was sent to France. There he saw a good deal of service at the front, serving part of the time as a litter bearer. In August, 1918, he was gassed and sent to a base hospital where he remained until returned to the United States in April, 1919. He was placed in a hospital at Camp Upton. There he was regarded as mentally disordered and sent to the psychopathic section. The examination there disclosed slow speech and difficulty in thinking. There was some tendency to worry about his family, but in general he was disinterested in his surround-

ings and showed little initiative. In June, 1919, he was sent to Ann Arbor. Here he showed a few abnormalities in appearance. His face had a perplexed and at times somewhat silly expression. In general, he was quiet and inactive. When urged to do a little work, his interest would soon flag and he would stand about until again started at something. His speech showed a number of peculiar mannerisms. In speaking he kept his lips rather tightly closed, and his responses were given in slurring, stuttering tones. The most notable feature of his stream of thought was the poverty of ideas. He spoke in short, choppy sentences. He complained of a drawing sensation in head. He heard his mother's voice telling him not to eat. He obeyed this, but afterward complained of the food tasting bad. At other times, he heard voices telling him pleasant things. He complained of the food hurting him mentally.

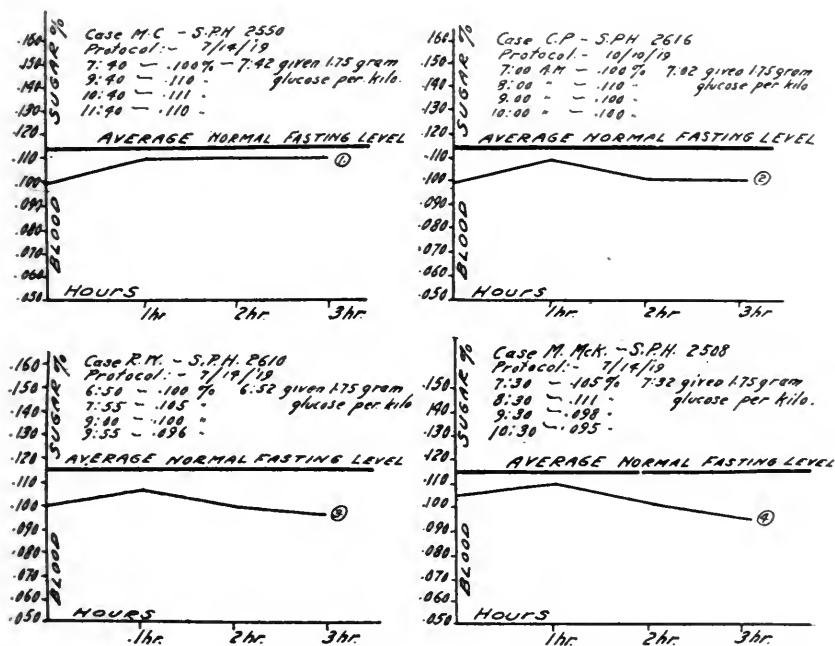


Fig. 5.—Individual sugar tolerance curves in manic-depressive insanity (hypo-manic group).

There were no important physical abnormalities. The chief neurologic symptoms were fine tremor of the tongue and fingers, and moderate dermatographia.

His condition continued without change until his discharge to his family on July 13, 1919.

CASE 14 (S. P. H. 2600).—Diagnosis: Dementia praecox, hebephrenic type (Fig. 3, Curve 7).

C. B., a man, aged 24, was admitted June 14, 1919.

His father was alcoholic and his mother neurotic. At the present time she has tics and choreiform movements.

At the age of 8 he is said to have had chorea. He was in school until he finished the eighth grade and was considered an average scholar. In temperament he was somewhat shy. At the age of 21, he had a neisserian infection. He worked as an unskilled laborer until June, 1917, when he entered in the

United States Army. The records of U. S. General Hospital No. 1 state that before he entered the Army he had a fear that if he were in action "he would get horribly wounded and suffer a lot." In France, this idea became more pronounced. He was gassed in October, 1918, and badly burned. After this experience he imagined there were holes in his lungs. He spoke of being dead and of having been in hell. He experienced visions in which he saw the devil. On the way back to the United States, he heard God's voice saying, "It's you, it's you." This he interpreted to mean that he was to save the world from a religious war. In General Hospital No. 1, he appeared dull and indifferent. His talk about his early life was fairly clear, but about recent experiences there was a marked incoherence. He laughed without provocation. At times, he was acutely hallucinated. He was transferred to the state psychopathic hospital, Ann Arbor, in June, 1919. Here he was quiet. He showed little interest in his ward surroundings. He never learned the names of his physicians or fellow patients. At times there was unmotivated laughter. He seemed a little apprehensive and talked with hesitation. His stream of thought was frequently interrupted by brief periods of blocking and abstraction. He complained of electricity being in his bed. He had hallucinations of hearing, but mentioned that the voices did not trouble him so much as formerly. There was usually a marked suggestibility, his hands and arms remaining where placed for considerable periods. At times there was flushing of the face.

The physical examination revealed, aside from the presence of a few casts in the urine, no important pathologic condition. The Wassermann reaction on the blood was negative. The neurologic abnormalities were fine general tremors of the extremities, widely dilated pupils and marked dermatographia of the skin surface.

His condition in the hospital remained unchanged until his transfer to a district hospital.

CASE 15 (S. P. H. 2410).—Diagnosis: Dementia praecox, paranoid type, apathetic deterioration (Fig. 3, Curve 8).

F. W., a woman, aged 20, was admitted Oct. 3, 1918. The family history showed that her father and grandfather had attacks of insanity. Until the age of 18 she was free from serious illness. At that age she entered a training school for nurses. She did not do well in her training course, and owing to peculiarities of conduct, she was ordered to leave. On returning to her home, it was observed that she was irritable, abstracted and forgetful. She expressed delusions of a persecutory type: that her family and others were against her and that she had not been properly cared for. Following this, she was admitted to this hospital Oct. 3, 1918, where her attitude showed a good deal of emotional tension. While she was obedient to the ward routine, she was restless and talkative. She related a long narrative, somewhat confused in detail, but in substance it was that she had been a sensitive, nervous child, who had not been understood by her parents, and this led to her becoming disordered in mind. She spoke of her head as being filled with wrong ideas all jumbled up. Everything that she had ever known was all mixed up. Voices talked to her and she had frightful visions. She appreciated that something was the matter and was glad to come to the hospital. During the further development of her condition in the hospital, she elaborated a phantastic story of having been a child born of noble parentage, who had been stolen away from her rightful home. She gave herself fanciful names and identified the physicians of the hospital by names of people who had played

a part in her various past experiences. There was always a perplexed, uncertain attitude toward what had happened to her and this led to frequent changes in her story. She became irritable when interviewed and kept by herself. She showed a progressive lessening of her interest in things about her. Auditory hallucinations became exceedingly troublesome. There were definite schizophrenic manifestations. She would remark: "Parts of my body become dislocated; sometimes I have blue eyes and sometimes I have black. Strange forms envelop me and control me." In the further development of

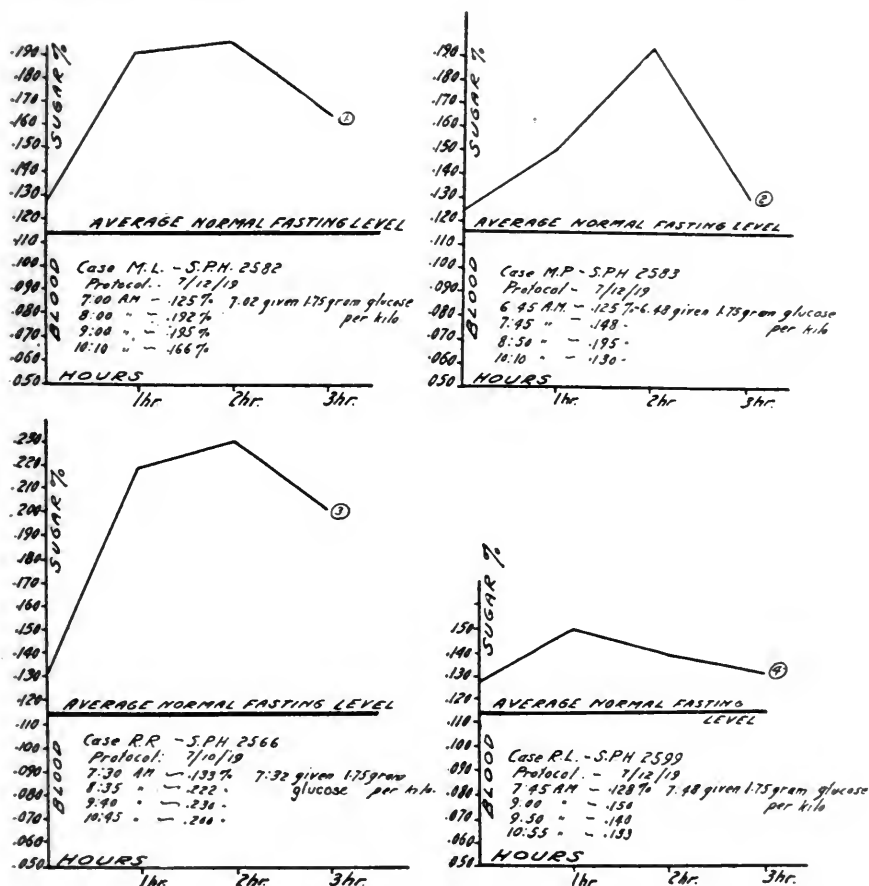


Fig. 6.—Individual sugar tolerance curves in manic-depressive insanity (depressive group).

the disorder her delusions became less coherent. Then developed a marked apathy. She lost all interest in her personal appearance. She kept by herself, lying for long periods of time on a couch or bed without speaking. At other times, she was more active and there were frequent outbursts of silly laughter. At the time of the tests her condition was one of apathetic stupor.

There were no important physical or neurologic abnormalities.

CASE 16 (S. P. H. 2592).—Diagnosis: Dementia praecox, catatonic type, negativistic stupor (Fig. 4, Curve 9).

M. K., a woman, aged 38, was admitted June 5, 1919. The family history shows that a sister had been insane. At about the age of 30, she developed an unusual interest in religious matters. Since the age of 34, she has been less efficient in her household work, and for the greater part of the time has been cared for by friends. During this period she expressed numerous somatic complaints of pain and numbness in her hands and body. About three months before admission, she had a brief attack of confusion in which she was disoriented and acted strangely. From then on, there was a more marked mental disorder. She expressed delusions of influence, spoke of the near approach of the day of judgment, saying that she had committed an unpardonable sin. She held conversation with imaginary people, and talked much of hypnotism. People were interfering with her thoughts. Her behavior varied between periods in which she would actively busy herself in housework and others in which she was inactive and mute.

On admission, she seemed to be in a confused, dreamlike state. She spoke of herself as being the wife of her former physician, and became muddled when she tried to explain how this could be. When she walked into the room she would go a few steps forward and then backward. At times her stream of thought showed very definite blocking. About two weeks later, she would sit in the same position for hours. Her feet would become cyanotic. For a considerable period she was absolutely mute. Threats produced no emotional response. This attitude was present during the period of the tests. At this time she held her body in a peculiar stereotyped attitude, paying no attention to anything going on about her. She actively resented passive movements and was mute. There were no serious physical or neurologic disturbances. The Wassermann reaction on the blood was negative.

CASE 17 (S. P. H. 2501).—Diagnosis: Dementia praecox, paranoid type, apathetic deterioration (Fig. 4, Curve 10).

T. G., a man, aged 37, whose family history was negative, until the age of 32 had been free from serious physical or nervous illness. At that time, he expressed delusions of suspicion that he was not fairly treated by his employers. This led to several changes of occupation. In August, 1918, he had a sudden attack of what was regarded as a heatstroke. In this, he was pale and "cold as ice." He returned to work the following week. Four days later he was found in his bathroom sobbing and crying. About a week after this he complained of people on the floor above bothering him with noises. He spoke of lights being flashed into his window. From this time on delusions of influence filled his thoughts and controlled his behavior. He had ideas that the form of his body had been changed. It became difficult for him to think coherently.

He was admitted here on Feb. 8, 1919. At that time he had clear comprehension and orientation, but his thought had an ambivalent quality. He expressed delusions of suspicion and reference. Many of his remarks were absurd. He spoke of eating sentences and words. When he bit into his food the sentences said things pertaining to his past life. Sometimes they gave directions as to what he was to do, saying, "Yes, no, yes, no, yes is no and no is yes." Auditory hallucinations were present more or less continuously for the first weeks of his residence. At times there were episodes in which he was negativistic. His delusions were unchanged. There developed rapidly an indifference to the hallucinations. At times there was unmotivated laughter. He usually kept by himself. At the time of the tests he was quiet, inaccessible

and showed little interest in his surroundings, but the delusions remained unchanged.

Physical and neurologic examinations revealed no serious abnormalities.

CASE 18 (S. P. H. 9).—Diagnosis: Dementia praecox, hebephrenic type, mild deterioration with desultory thought and impulsive conduct (Fig. 4, Curve 11).

C. C., a man, aged 26, was admitted Feb. 7, 1906. The family history was negative. At the age of 22, while a student in college, he became unable to study. He complained of his head getting light on the inside, and of being unable to grasp even the simplest ideas. He was removed from school, but continued to show mental peculiarities. He became seclusive and explained his

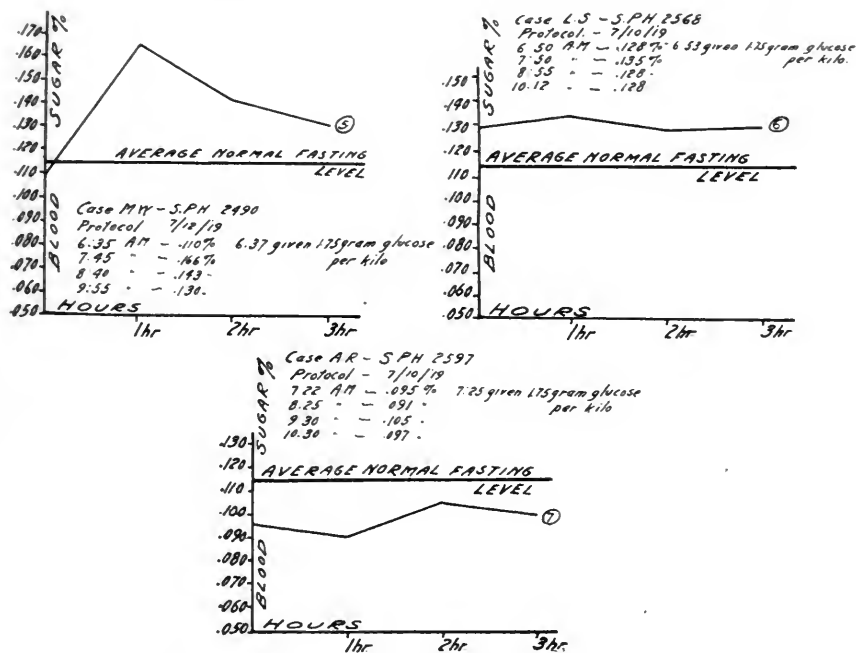


Fig. 7.—Individual sugar tolerance curves in manic-depressive insanity (depressive group).

difficulty at school as having been due to being shot through the head. Several months later, he was given employment in a broker's office. After about six weeks he was dismissed because of the mistakes he made. He was allowed to return to college, but soon disappeared. He went to Chicago where he behaved strangely. He expressed many expansive ideas, declared himself to be a monarch, attempted to interview prominent people, entered a private home and was apprehended by the police. He was placed in a private hospital where he continued to express expansive ideas and often laughed with unknown cause. His conversation became less coherent. He gave wrong names to those around him. This condition continued with little change until 1906 when he was transferred to this hospital. From then until the present time, thirteen years, there has been little change in his condition. He has continued clear in comprehension, orderly in conduct and extremely helpful about

the hospital. He is impulsive and somewhat irritable. At times he betrays a confused content of thought in which are mingled fragments of former delusions and falsifications of realities.

At no time has he shown any serious physical or nervous disorder.

MANIC-DEPRESSIVE GROUP

In this group are included eleven cases. Of these four were quite typically of the hypomanic phase, and Cases 1, 2, 4, 5 and 6 were in the depressed phase. Two cases are included here which probably belong in this group, but yet were somewhat atypical in their clinical symptomatology. Case 3 presented features suggesting the influence of a toxic state with definite symptoms of unclear consciousness, and Case 7 showed an unusual prominence of anxiety. Both of these patients showed types of curves varying from the remainder of the group.

HYPOMANIC GROUP

This group includes four cases. The data and curves are given in Figure 1*B*, Curves 1 to 4 and Figure 5, Curves 1 to 4.

CASE 19 (S. P. H. 2550).—Diagnosis: Manic-depressive insanity, hypomania, second attack (Fig. 5, Curve 1).

M. C., a woman, aged 49, was admitted April 16, 1919. The family history was bad. The father died of apoplexy; one brother was insane, one sister had been insane and a suicide. The patient's first attack occurred at the age of 27, coming on after the suicide of her sister. For six weeks she was mildly overactive, irritable, euphoric in mood and was unable properly to care for her household duties. She made a good recovery and continued well until the age of 49. Then worrying over the absence of her son in military service, she became overactive, wrote numerous letters and busied herself unduly with the affairs of others. Since January, she has had recurring periods of mild excitement, at intervals of about eight days and followed by a mild depression lasting about two days. On admission here she was mildly overactive, talked much, and wrote many letters, and was usually a little exhilarated in mood. Usually she was extremely distractible and her stream of thought often gave definite flights of ideas. Her reactions were characteristically those of a mild hypomania.

There were no important physical or neurologic abnormalities. After three months' treatment, she had improved greatly and returned to her home.

CASE 20 (S. P. H. 2616).—Diagnosis: Manic-depressive insanity, hypomania, with history of several previous attacks (Fig. 5, Curve 2).

C. P., a man, aged 53, was admitted July 11, 1919. The family history was negative. At the age of 37, he had his first attack of mental disorder. At that time he was overactive, sleepless and unable to concentrate his interest on his work. After a few months he regained his normal level. Similar attacks have recurred about every two years, lasting from three to six months. The present attack began in March, 1919. For about three months he was inactive, complained of being tired and slept poorly. This gradually passed into a phase of overactivity with euphoric mood, distractible attention, marked talkativeness and clear comprehension and orientation. In this condition, he

was admitted to this hospital. Here he has been overactive, distractible, boastful, with marked flight of ideas. His mood has been constantly one of elation.

His physical and neurologic examinations showed no serious abnormalities.

CASE 21 (S. P. H. 2610).—Diagnosis: Manic-depressive insanity, hypomania, with marked delusional elaboration (Fig. 5, Curve 3).

R. W., a man, aged 23, was admitted, June 30, 1919. The family history showed environmental difficulties, but no insanity. Following a fracture of the leg at the age of 22, it was observed that his disposition had changed. He was more irritable and quick tempered. Two weeks previous to admission he expressed delusions of persecution and became apprehensive that the police were after him. There progressed from this time an increasing excitement with excessive talkativeness and delusions of reference and persecution. On admission here, he was overactive, irritable and talked much in a boastful way. He was always clear in comprehension. His attention was distractible. The stream of thought showed a characteristic manic flight of ideas. His mood was irritable and expansive. His content of thought was intermixed with ideas of reference and erotic phantasies. At the period of the tests he was overactive and showed a marked flight of ideas and elevated mood.

There were no physical or neurologic abnormalities. The Wassermann reaction on the blood was negative.

CASE 22 (S. P. H. 2508).—Diagnosis: Manic-depressive insanity, hypomania, with paranoid reactions (Fig. 5, Curve 4).

M. McK., a woman, aged 31, was admitted June 28, 1919. Aside from alcoholism of the father, the family history was negative. She was married at 17 and had had five pregnancies. Several of these were premature births and one was a miscarriage. In 1916, a definite change was observed in her mental attitude. She became more irritable and expressed ideas of a paranoid quality. She spoke of herself as having unusual ability and of being able to influence others around her. A few months later, she wrote letters filled with peculiar expressions of profane or religious character. They were written in a hand writing different from the one she usually used. She expressed delusions regarding her neighbors and accused them of impossible sex immoralities. In April, 1919, she had an attack of influenza and following this her mental abnormalities became exaggerated. On admission here she had numerous complaints of physical discomforts. She was restless, talked much, with a tendency toward distractibility. Her mood was usually euphoric with occasional impulsive outbursts of irritability. The content of her thought had a paranoid trend, with ideas of reference and false interpretations of her experience in the hospital. There was no distinct flight of ideas, but her speech reactions had a definite manic quality. There were no hallucinations and aside from occasional remarks showing an ambivalent quality there were no schizophrenic symptoms.

Physically, she had mitral stenosis with regurgitation that was well compensated. The Wassermann reaction of the blood was negative. There were no neurologic abnormalities.

DEPRESSIVE GROUP

This group includes seven cases. Two cases are included here which probably rightly belong here, but yet were somewhat atypical in their clinical symptomatology. Case 3 presented features suggesting

the influence of a toxic factor with definite symptoms of unclear consciousness. Case 7 showed an unusual prominence of anxiety. Both of these cases showed types of curves varying from the remainder of the group. The data and curves are given in Figure 1C, Curves 1 to 7, and in Figures 6 to 7, Curves 1 to 7.

CASE 23 (S. P. H. 2582).—Diagnosis: Manic-depressive insanity, depressed phase. Emotional depression with psychomotor retardation and mild delusional elaborations (Fig. 1 C, Curve 4; Fig. 6, Curve 1)).

M. L., a woman, aged 28, was admitted May 26, 1919. Aside from insanity of a grandparent, the family history shows no definite mental disorder. The patient has always been of a nervous temperament which prevented her from successfully completing her school period. At 23 she married, and for a brief period she had a mild depression. This passed off and she continued in her usual health until December, 1918, when she entered gradually on a period of mental depression. At first, this showed itself in feelings of inadequacy and worry about her domestic responsibilities. There then developed ideas of self-accusation and personal unworthiness, which continued until her admission here. Her attitude then was one of deep depression. She was inactive, moved slowly and spoke only when urged. Her replies were given in low tones and short sentences. At times her content of thought showed delusions of negation. She insisted that she had never attended school; that her father and mother were dead; that those about her were not real people; that the food contained disgusting substances, and that the nurses and physicians did not treat her properly.

There were no serious physical or neurologic abnormalities.

CASE 24 (S. P. H. 2583).—Diagnosis: Manic-depressive insanity. Emotional depression with retardation and delusions of unworthiness (Fig. 1 C, Curve 1; Fig. 6, Curve 2).

M. P., a woman, aged 36, was admitted May 27, 1919. The family history shows that her grandfather committed suicide. Her mental disorder appeared seven months before admission, when following the death of her brother she became depressed. The death of another brother was accompanied by a deepening of her depression with several serious efforts at suicide. On admission, she was deeply depressed. Her stream of thought was greatly slowed. Her content of thought was one of sadness with ideas of unworthiness. She spoke but little, and there was little spontaneous activity. There were no hallucinations nor evidences of schizophrenic disturbances. There were no serious physical or neurologic abnormalities.

CASE 25 (S. P. H. 2566).—Diagnosis: Manic-depressive insanity, atypical depression, with marked prominence of hallucinations, and episodes of unclearness, following influenza (Fig. 1 C, Curve 3; Fig. 6, Curve 3).

R. R., a woman, aged 37, was admitted May 5, 1919. The family history was negative. Her early life was unimportant. She was married and had eight children. No mental disorder had been noted until December, 1918, when she had influenza, during which she was delirious. With her physical improvement, her mental disorder disappeared to the extent that she took care of her home for about six weeks, when there occurred a sudden change. She became depressed and inactive; made serious attempts at suicide and threatened to kill her children. On admission, she was confused and stuporous. Her stream of thought was slow, and when fatigued she became confused in her responses..

There was little spontaneous speech. Her attitude continued depressed with marked retardation of the stream of thought until September 12, when she suddenly became excited. There were vivid auditory hallucinations to which she reacted with great irritability. A voice came from the clock threatening her and her children. Since then her excitement has lessened, but the hallucinations have continued. She now holds conversations with these. Her affect toward these is less keen than formerly. At the time of the tests she was clear in her understanding of questions and in orientation. The hallucinations were active and at times there were impulsive outbursts of brief duration provoked by the voices which were calling her vile names.

The physical findings at admission were poor nutrition, blood pressure: systolic, 138; diastolic, 90. The Wassermann reaction on the blood was negative. There was no pathologic condition of the organs of the body. There were no definite neurologic findings.

CASE 26 (S. P. H. 2599).—Diagnosis: Manic-depressive insanity, depression with marked retardation (Fig. 1 C, Curve 4; Fig. 6, Curve 4).

R. L., a man, aged 30, was admitted July 1, 1919. While a soldier overseas, he developed a mental disorder which brought about his admission to a base hospital. His attitude while there was described as being one of depression. He spoke but little and then in low tones. He was returned to this country and for a brief period was in an army hospital. There he continued to be depressed and unproductive. He was transferred to this hospital on July 1, 1919. Here he was restless, pacing about his room for hours. He rarely spoke in audible tones. He understood questions clearly and obeyed requests correctly. His stream of thought was extremely slow, as if retarded. There were no delusions or hallucinations.

There were no serious physical or neurologic findings and the blood Wassermann reaction was negative.

CASE 27 (S. P. H. 2490).—Diagnosis: Manic-depressive insanity, depression of the involution period with hysterical symptoms (Fig. 1 C, Curve 5; Fig. 7, Curve 5).

M. W., a woman, aged 45, was admitted Jan. 31, 1919. The family history shows that her mother has had periodically recurring attacks of depression since the menopause. The patient had undergone three surgical operations: one for peroneal repair following the birth of her first child, one for hernia a few years ago, and a third for chronic appendicitis in June, 1918. At that time she had been complaining a good deal of indefinite abdominal pains, which were not relieved by the operation. She became apprehensive about the effect of the operation. She soon expressed delusions of being commanded by the Lord to fast. She refused food for a considerable period of time, and on Jan. 31, 1919, she was admitted to this hospital. She was physically feeble and remained in bed for a number of weeks. Her attitude was characterized by numerous complaints about her physical state. She would not eat because the food stuck in her throat. She was inactive, talked but little and was continuously depressed and worried. She was fed mechanically for a number of days. She expressed a variety of delusions. The food had kerosene in it. The food she ate remained in her stomach. Her bowels did not move. She heard voices talking to her at night; her little girl called to her. She received warnings not to eat. It was wrong for her to eat. By doing this she had brought trouble on her family. In April, her depression was less marked. She was more active and expressed her delusional ideas less frequently. Her

improvement continued with great gain in weight until her discharge in September, 1919. The physical examination revealed subjective feelings of weakness. Her weight was 112 pounds. The most notable abnormality was an extremely rapid heart. The pulse rate was 132. There were no definite murmurs. Her blood pressure was 104 systolic and 86 diastolic. The urine and blood were not pathologic. The Wassermann reaction on the blood was negative. Her menstruation had been irregular for several months. Both pupils were slightly irregular. The conjunctivae were anesthetic. The left epigastric and abdominal reflexes were absent. Her improvement continued with gain in weight until her discharge in September, 1919. While her depression had in a large measure passed away, she was markedly abulic and showed little spontaneous interest in her surroundings.

CASE 28 (S. P. H. 2568).—Diagnosis: Manic-depressive insanity, emotional depression with marked retardation (Fig. 1 C, Curve 6; Fig. 7, Curve 6).

L. S., a woman, aged 53, was admitted May 9, 1919. The family history was negative. Until ten months previous to admission, she had shown no nervous or mental disorder. At that time she became apprehensive that an herpetic eruption would become cancerous. She worried much over this and consulted many physicians. In spite of their assurances that her fears were needless, she became more depressed. Following the illness of her son, she slept badly and ate but little. While in a private hospital she became apprehensive during an operation in a neighboring room and cut her wrist with a piece of glass. Following this, she had the fear that a clot had formed on her brain as a result of having cut her wrist. She imagined that her features had changed, that she would lose her eye sight, and at night she feared to close her eyes lest she should never see the family again. About March 1, she expressed the delusion that her stomach was so full that she could not eat. There had for some time been developing a gangrenous condition of the great toe. At her admission here, she was deeply depressed and extremely apprehensive. She rarely spoke and remained inactive. There was little productivity of thought. Occasionally she expressed delusions of unworthiness or somatic complaints. She resisted the attentions of nurses and ate only on urging.

She was 5 feet, 1 $\frac{3}{4}$ inches in height and weighed 107 pounds. She was poorly nourished and feeble in strength.

There were no serious abnormalities of the body organs. The blood pressure was 150 systolic, and 90 diastolic. The Wassermann reaction on the blood was negative. The urine was not abnormal. The right great toe was gangrenous. This slowly improved during her residence and finally entirely recovered. Aside from tremors of the tongue and hands there were no neurologic findings. There was no improvement in her mental condition and her weight had fallen considerably at the time of her removal from the hospital on Sept. 3, 1919.

CASE 29 (S. P. H. 2597).—Diagnosis: Manic-depressive insanity, depression with agitation (Fig. 1 C, Curve 7; Fig. 7, Curve 7).

A. R., a woman, aged 32, was admitted June 11, 1919. The family history was negative so far as known. Temperamentally the patient was nervous and easily excited. Feb. 2, 1919, she gave birth to a child. About two weeks later she underwent a laparotomy for some unknown abdominal pathologic condition. Two weeks after this she accidentally fell from a chair and from then on she began to be very anxious concerning herself. Various somatic delusions were expressed. She complained of heart trouble, consumption and various physical

ailments. Her delusions soon took on a religious character. The wrath of God had come upon her. She belonged to the devil. There were numerous self-accusations and ideas of unworthiness. She twice attempted to kill her husband and made one serious attempt upon her own life. On admission here, her mood was one of depression with agitation. While in bed she was restless and picked at her fingers and face. While up she walked restlessly about the ward in a tense anxious manner. Her content of thought was one of ideas of unworthiness and self-accusations for her past conduct.

Physically, there were no serious abnormalities. The blood pressure was 119 systolic, and 78 diastolic. The Wassermann reaction on the blood was negative. The neurologic examination showed dulling of pain appreciation on the anterior surfaces of the body and extremities. The tendon reflexes were somewhat diminished. The fingers on extension were tremulous.

DISCUSSION

Referring to the group of normal curves on the composite chart, we note at a glance that these seven curves are practically parallel throughout. Further, we note that the average initial fasting level is about 0.115 per cent., and that the acme level varies considerably, but in every case the primary level is reached within two hours. The average normal fasting level was established on normal persons, carefully controlled as mentioned above. When so controlled, we find that we get a higher average than when no attention is paid to rest and the general condition of the patient. Nevertheless, we do not mean to establish this as a fixed level. Our primary purpose is to use this more or less arbitrary level as a better means for comparison of the curves. We wish to lay no great stress on the normal initial fasting levels, or the acme to which they arise, but on the other hand, we do wish to emphasize the value of the general shape of the curve and the relative zone, that is, above or below the fasting level, on the chart in which the group falls.

In comparing our curves with those obtained by Kooy,⁵ it must be borne in mind that he uses considerably less carbohydrate in his tolerance test than we have. We emphasize the fact that the acme level, which Kooy regards as of much importance, varies a great deal among the normal and still more among the abnormal subjects. Among our cases, the acme level seemed to tell us very little.

Reference to the individual curves and to the composite chart seems to reveal that as a group the manic depressive patients who are in the depressed phase have a suggestion of an initial hyperglycemia. The rise is distinctly more pronounced than in the normal, and the elevation is maintained well beyond the second hour, that is, the tolerance seems definitely delayed. The tolerance curves of these patients, excepting Case 7, which we mentioned as being atypical in its symptoms and course, have a diabetic character, the degree of which varies with the clinical status of the patient. The curve in Case 7 has

similarities to that of the hypomanic group. The symptoms, however, were predominantly those of an extreme degree of depression with great agitation. It was a type of case not uncommonly encountered in which the clinical position is usually placed among the manic-depressive psychoses.

Case 3 was also atypical in its clinical course. Its close association with an infectious disease and the occurrence of a lack of clearness of consciousness and other features characteristic of toxic psychoses, might suggest that it did not belong in this group. There was, however, a persistent depression and a psychomotor retardation rather characteristic of manic-depressive cases. Its curve is of the general type of the other cases of the group, but of far greater height.

The cases in the hypomanic phase showed a tolerance curve that was almost flat, when compared with the cases of any of the other groups including the normal. The initial reading is quite low, in fact, slightly below the normal average.

The curves of the dementia praecox group are strikingly different from those of the others. They show a great variation in their acme levels; yet, the shape of all curves within the group is practically the same. Curves 1 to 7, inclusive, are all practically parallel to one another. All of these patients were in the acute phase. The general shape of the curve differs from the normal in that the initial fasting level is lower, the acme is relatively high, and the return to the primary level takes more than three hours, there being a very definitely delayed tolerance.

Curve 11 of this series is perfectly normal in its height and shape. It is of interest to note that this is a case of about seventeen years' duration. The first two years of the course were characteristically those of an acute phase of dementia praecox. At the end of that time, he reached a very satisfactory adjustment and for the last fifteen years has remained stationary with a very mild degree of deterioration.

Curves 8 and 9 bear the general shape of the others of this group, but the acme is much higher. Clinically, both of these cases stood out from among the others of this group on account of certain special features. In Case 8, there were marked variations in the affective reactions. At times, there were periods of extreme confusion and perplexity with marked disturbance of association, at other times periods of silliness or apathetic stupor. Case 9 was one of catatonia with negativistic stupor.

Curve 10, which also varied from the majority of those of the group, was clinically somewhat different. There had for a long time been no acute manifestations, his attitude being one of quiet apathetic deterioration.

Consideration of the curves reveals at once that the acme level of the curve is of no special diagnostic value. While cases of the dementia praecox group and of the depressed phase of the manic depressive group have the same acme level, there is a striking difference in the shape of the curves of the two groups. It is the general topography of the curve rather than any one point that we consider of differential diagnostic importance among the groups studied. Whether or not all of the variations of these curves can be explained by the emotional reactions of the patients, this in turn being in interrelation with a hypersecretion of epinephrin, thus mobilizing the glycogen of the liver and hence increasing the amount of glucose in the circulatory blood, is an unsettled question. Stewart and Rogoff⁷ have proved that asphyxia and narcosis cause hyperglycemia, and that this exists after the suprarenals have been removed, and further, that the glycogen store of the liver is unaltered. To what extent this may have been influenced by other parts of the chromaffin system that may not have been removed is undetermined.

When the curves of this series are analyzed, it is noticeable that many of them have a prolonged acme level far above the normal threshold when sugar appears in the urine, yet in only one case was sugar found in any of the urines of this series. This was in Case 3 of the group of depressions, when the height and duration of the acme level would lead one to anticipate a glycosuria.

CONCLUSIONS

It may be stated on the basis of the data secured in these experiments, employing the Benedict modification of the Lewis-Benedict method for blood sugar, that tolerance curves differ from those obtained in normal individuals, in cases of the hypomanic and depressed phases of manic depressive insanity and in cases of dementia praecox; that among the pathologic groups studied there were striking differences among the different groups; that in each clinical group, the curves had features in common that suggest a type curve; that among cases of dementia praecox, tolerance curves vary according to the phase of the clinical course.

7. Stewart, G. N., and Rogoff, J. M.: Relation of Adrenals to Certain Experimental Hyperglycaemia (Ether and Asphyxia), *Am. J. Physiol.* **51**:366 (March) 1920.

THE SEROLOGY OF THE SPINAL FLUID AND BLOOD IN EPIDEMIC ENCEPHALITIS*

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The subject matter which we present has, for the sake of clearness, been divided into three parts; the first contains a review of the literature, the second, data based on the 245 case reports obtained from the foregoing sources, and the third, a section devoted to differential diagnosis by means of the blood and spinal fluid serology.

REVIEW OF THE LITERATURE

Until the publication, in 1917, of the description of the first cases of epidemic encephalitis in Vienna, no reliable statistics of the changes in the blood and spinal fluid in this disease are obtainable. At this time, von Economo¹ gave an account of the clinical and laboratory findings which we shall consider the first accurate data on the subject. He pointed out the following changes in the spinal fluid: An excess of globulin was found and the spinal fluid cell counts ranged from normal to 43. The fluid was clear and, in some cases, under pressure. The only group of observations which were not mentioned by von Economo concerned changes in the quantity of sugar in the spinal fluid. His observations on the colloidal gold curve were summed up in a sentence. No curves were given. In this country, curves were first published by Josephine B. Neal² in September, 1919. She mentioned the abnormal changes and the similarity to those curves found in acute anterior poliomyelitis. Following this, observations on the occurrence of syph-

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* The material for this article was obtained from records of certain New York Hospitals: Bellevue, Mt. Sinai, The Neurological Institute, New York, Presbyterian, Roosevelt and St. Luke's, and from the following physicians: James B. Ayer, Boston; Lewellys F. Barker, Baltimore; Peter Bassoe, Chicago; Josephine B. Neal, New York; Harry C. Solomon, Boston; Walter F. Schaller, San Francisco; E. W. Taylor, Boston, and Lloyd J. Thompson, Boston.

We are extremely grateful to the chiefs of service of these hospitals and to the individual physicians who have sent us copies of their records.

1. Von Economo: Encephalitis Lethargica, *Wein. klin. Wchnschr.* **30**: 581, 1917.

2. Neal, Josephine B.: Lethargic Encephalitis, *Arch. Neurol. & Psychiat.* **2**:271 (Sept.) 1919.

ilitic and paretic types of curves were made by Bassoe,³ Brill and Benson,⁴ Davis and Kraus,⁵ and Archambault.⁶

The foregoing gives an idea of the chronological order of the description of the spinal fluid findings. The following paragraphs will be devoted to a detailed account of the spinal fluid and blood findings given in the literature.

A. SPINAL FLUID: *Appearance.*—In the greatest majority of cases, observation records a clear, colorless fluid. Bloody fluids have occasionally been found, in fact more commonly than could be accounted for by the puncture of a vein. A xanthochromic appearance has been extremely rare.

Pressure.—The pressure of the spinal fluid varies from normal to a considerable increase.

Cell Counts.—In spite of the fact that von Economo in his original description made note of a pleocytosis, the French, who had studied the matter carefully during the years 1918-1919, had come to the conclusion that a normal cell count was a rule and of diagnostic importance. It was not until Dec. 10, 1919, when Bénard⁷ reported a case with pleocytosis, that doubt was thrown on the invariability of normal spinal fluid cellular content in epidemic encephalitis. The absence of pleocytosis before this date, as noted by the French, was not found by the British and American observers. The latter published cases seen in 1919, in which there appeared more than the normal number of cells. The cells which appeared in the spinal fluid were always predominantly of the mononuclear type although polymorphonuclear increase up to 15 per cent. had been found in many cases.

The impression gathered from the literature is that the number of cells may vary from normal to an increase of several hundred.

Globulin.—The discrepancy between the report of the French, who noted an absence of globulin in the early cases of the epidemic, and the English and Americans who had reported its presence in increased

3. Bassoe, P.: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

4. Brill and Benson: Lange Reaction in Epidemic Encephalitis, J. Lab. & Clin. Med. **5**:113 (June 20) 1920.

5. Davis and Kraus: The Colloidal Gold Curve in Epidemic Encephalitis, Am. J. Med. Sc. **161**:109, 1921.

6. Archambault, La Salle: Choreo-Athetoid and Choreopsychotic Syndromes as Clinical Types or Sequelae of Epidemic Encephalitis, Arch. Neurol. & Psychiat. **4**:484 (Nov.) 1920.

7. Bénard, R.: Le liquide cephalo-rachidien dans l'encephalite léthargique, Paris méd. **10**:474 (June 5) 1920. (This article contains a thorough review of the French literature up to June, 1920. References from the literature of other countries are also given.)

amounts, is a parallel to the reports on the cell counts. In general, it may be said that an increase in globulin occurs in the majority of cases.

Sugar.—No report of the amount of this substance was made until the end of 1919. From that time until the present, the French have published fifteen observations on the amount of this substance in the spinal fluid. These varied from 0.067 per cent. to 0.106 per cent, averaging 0.085 per cent. It would seem, therefore, that the amount of sugar in the spinal fluid, wherever it has been reported, has exceeded the normal quantity.

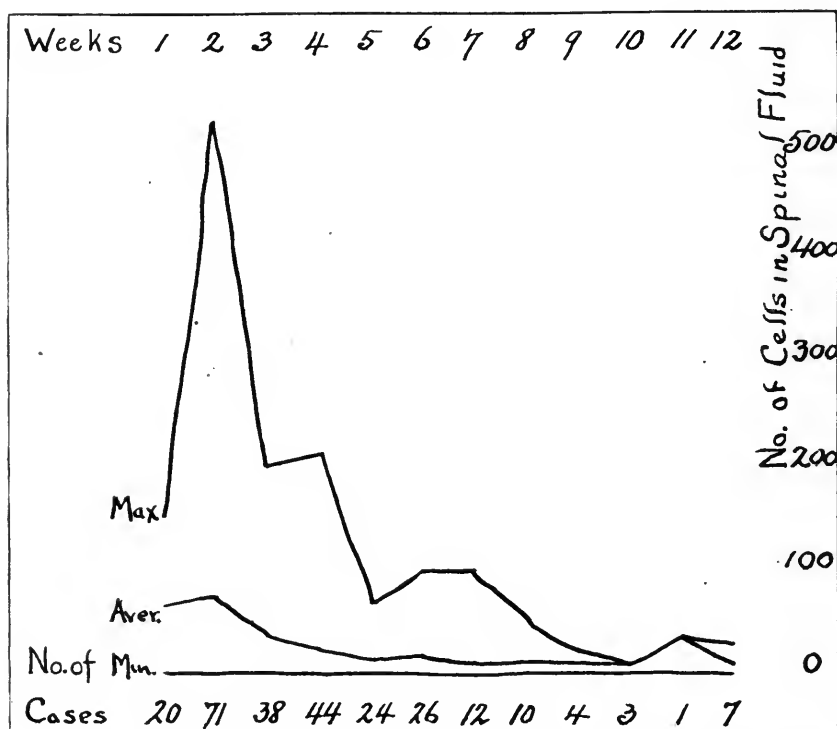


Chart 1.—Variation in the number of cells in the spinal fluid.

Wassermann Reaction.—This has been found negative in the spinal fluid whenever it has been reported except in syphilitic cases.

Colloidal Gold Reaction.—Von Economo¹ commented briefly on the reaction of colloidal gold solutions as follows: "The gold-solution reaction shows no typical elevation of its curves." This implies that some changes existed.

B. BLOOD: The white blood counts, as reported in the literature, reveal a variation from 3,000 to 25,000, the average being about 15,000. Slight polymorphonuclear increase is the rule. The remainder of the

blood picture is normal. Observations on the blood chemistry have been scattered and incomplete. The blood Wasserman reaction is reported negative.

THE EVIDENCE OF CASE REPORTS

The general statistics in Table 1 give a numerical summing-up of the case reports on which we base our data.

TABLE 1.—NUMERICAL SUMMING UP OF CASE REPORTS

Number of cases	245
Number of spinal fluid cell counts	260
Number of spinal fluid globulin determinations	214
Number of spinal fluid sugar determinations	12
Number of spinal fluid colloidal gold curves	120
Number of spinal fluid Wassermann tests	185
Number of blood Wassermann tests	161
Number of blood polymorphonuclear counts	171
Number of blood differential counts	153

A. SPINAL FLUID: As a rule, the spinal fluid shows considerable and important changes in epidemic encephalitis. These are extremely variable, not only in different cases, but also in the course of each case. The most characteristic changes are:

1. A clear, colorless fluid.
2. With an increase in the pressure of the cerebrospinal fluid when withdrawn.
3. An increase in the number of mononuclear cells.
4. An increase in the amount of globulin.
5. An increased amount of spinal fluid sugar.
6. A negative Wassermann reaction.
7. The presence of bodies producing a change in the color of a solution of colloidal gold (Lange reaction).

1. *Appearance*.—The fluid is, in most cases, clear and colorless. Occasionally bloody fluid is obtained.

2. *The Pressure*.—As would be expected, the pressure of the spinal fluid is sometimes very much increased. Here also no rule exists, and, though this hypertension is frequent, it is not universal.

3. *The Cell Count*.—Table 2 indicates the minimum, maximum and average number of cells during each of the first twelve weeks and during the fourth to twenty-fourth months of the disease. The graph in Chart 1 presents this in a different way. It may be seen that, on the average, more cells are found during the first three weeks of the disease. The value of this in individual cases is diminished by the variations in the number of cells in each week. An idea of this may be had from an examination of the columns showing maximum and minimum number of cells for each week and from Table 3. The number of polymorphonuclear cells may be as great as 15 per cent.

Fluctuation in the number of cells occurs during the course of the disease. When there is a remission, the cell counts may increase.

4. *Globulin*.—The globulin is usually increased to a moderate degree (in 72 per cent. of the cases). However, since this is not always true, its diagnostic value in individual cases is lessened. The presence of globulin is not always paralleled by the presence of cells or a positive colloidal gold curve.

5. *The Sugar Test*.—The majority of the tests have been done at Bellevue Hospital since May, 1920. No estimates were given in case reports outside of New York.

TABLE 2.—NUMBER OF CELLS IN THE SPINAL FLUID

Weeks	Minimum	Average	Maximum	Cases
1	2.0	63.3	160.0	20
2	0.0	71.0	540.0	71
3	0.0	37.3	200.0	38
4	0.0	28.2	222.0	44
5	1.0	21.3	70.0	24
6	0.0	25.7	100.0	26
7	1.0	21.0	101.0	12
8	2.0	16.2	48.0	10
9	0.0	13.0	32.0	4
10	5.0	8.0	14.0	3
11	4.0	40.0	40.0	1
12	2.0	15.2	33.0	7
Months				
4	4.0	40.2	108.0	9
5				
6	3.0	32.0	61.0	2
7		2.0		1
8	2.0	2.5	3.0	2
9				
10				
11				
12				
13				
21		2.0		1
24		3.0		1

The number of estimations is insufficient to enable us to draw any but tentative conclusions. However, those figures which we have show, without exception, values above normal.

The sugar content of the spinal fluid has been tested in twelve cases. It has ranged from 0.062 per cent. to 0.095 per cent. (Table 4).

6. *The Wassermann Test*.—This was negative when reported.

7. *The Colloidal Gold Curve*.—This has been found changed in 100 out of 120 cases (83 per cent.). The type of curve varies considerably, as Chart 2 shows. There is a tendency toward elevation of the left-hand part of the curves in the later stages of the disease, but this is not constant. Positive curves were found as late as the twenty-first month. Elevations of the right end of the curve alone were not found.

TABLE 3.—NUMBER OF CELLS IN THE SPINAL FLUIDS AT VARIOUS WEEKS INCLUDING REPETITION OF CELL COUNTS*

1	2	3	4	5	6	7	8	9	10	11	12
4	—	—	5	—	—	—	—	2			
100	—	—	—	—	—	—	15				
70	30	—	—	60	70	—					
100	—	—	—	—	8						
5	10	—	5								
3	—	20									
40	—	4									
50	—	4									
160	90										
160	64										
9											
5	21	38	28	20	—	—	15				
5	8	10	—	8							
9	{180}	—	—	45							
11	{350}										
25	19	18	32								
26	100	40	40								
120	{130}	—	26								
150	{540}										
150	270		149								
150	{15}	25									
	{22}										
	6	20									
	7	7									
	16	18									
	{22}	{16}									
	{36}	{43}									
	37	7									
	44	2									
		12									
	46	6									
	150	200									
	4	6									
	0										
	0	100	—	—	—	—	18				
	3										
	4										
	4	0									
	5	0	0	—	—	—	2				
	5	2	0	—	12	—	8				
	7	5	150	—	13						
	7	6	20	15							
	10	10	5	10							
	10	12	43	{19}							
	12	114		{36}							
	12	18									
	12	18									
	14	20	0								
	18	30	0	3	—	3					
	20	31	6	30	—	6					
	20	48	6	30	{40}						
	122	{50}	6		{42}						
	25	{75}	6		{46}						
	27	60	6		{57}						
	29	104	7								
	{30}	134	{8}	1							
	{100}	170	{11}	4	97	—	—	—	—	—	33
	30		{8}	4	48	14	14				
	30		{15}	5							
	43		9	8	0						
	45		10	9	0	3	—	—	5		
	50		{10}	15	0	22	—	—	14		
	{54}		{66}	16	0	30	48	32			
	{160}		{78}	16	{2}						
	56		11	21	{11}	1					
	70		15	28	{12}	3	10	—	5		
	71		18	40	4	11					
	72		24	70	5	28	2				
	80		25		7	30	30	0			
	108		28		13	101		5			
	{190}		30		100						
	{230}		30								
	200		35								
	270		35								
	650		40								
			66								
			95								
			222								

* Except where repetitions occur, the figures are arranged in an ascending order. Bracketed figures indicate several observations on the same case and in the same week.

The tendency is to a color change in the high and medium concentrations of spinal fluid. Changes in the low concentrations do not occur alone, but may occur when the medium and the high concentrations are altered.

B. BLOOD: Normal values for red blood count and hemoglobin have invariably been found. Leukocyte counts have varied from a minimum of 4,500 to a maximum of 32,000, the general average being 12,000. Chart 3 shows these relations in graphic form.

TABLE 4.—SUGAR CONTENT OF SPINAL FLUID IN TWELVE CASES

Week	Month	Percentage of Sugar in the Cerebrospinal Fluid
3	..	0.082
3	..	0.075
6	..	0.068
9	..	0.062
9	..	0.094
..	4	0.065
..	4	0.080
..	4	0.094
..	4	0.092 *
..	5	0.070 *
..	5	0.063
..	10	0.095

* Observations on the same case.

Maximum, minimum and average percentage of polymorphonuclears are shown in the graph in Chart 4. This will serve to emphasize the extent of the variations in the various stages of the disease.

The number of observations on the chemistry of the blood are too few to enable us to draw any conclusions.

TABLE 5.—PERCENTAGE OF SUGAR IN THE BLOOD IN TWELVE CASES

Week	Month	Percentage of Sugar in the Blood
2	..	0.095
2	..	0.75
3	..	0.100
3	..	0.100
3	..	0.111
3	..	0.083
4	..	0.120
7	..	0.100
..	4	0.110 *
..	5	0.170 *
..	6	0.143
..	7	0.101

* Observations on the same case.

A few observations (12) on the amount of sugar in the blood have been recorded and are within normal limits. They are recorded in Table 5.

Wassermann Reaction.—The Wassermann reaction has been found negative when reported.

DIFFERENTIAL DIAGNOSIS

1. Other Forms of Meningitis: (a) serous, (b) acute purulent, (c) tuberculous, (d) syphilitic meningitis and neurosyphilis.
2. Acute Anterior Poliomyelitis.
3. Brain Tumor.
4. Parkinson's Syndromes and Allied Conditions.

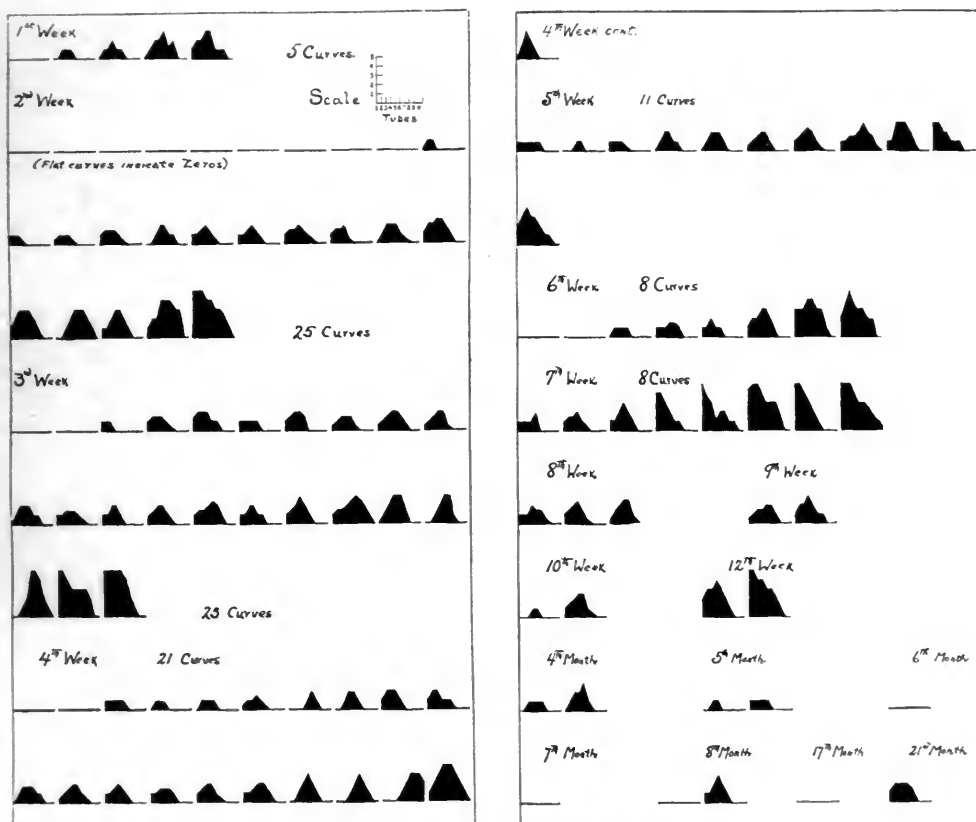


Chart 2.—Colloidal gold curves.

5. Multiple Sclerosis.
6. Polyneuritis.

In order to use the serology of the spinal fluid in epidemic encephalitis for the purpose of differential diagnosis, it is necessary to make observations on the pressure, the cell count, the globulin, the presence of sugar, the colloidal gold curve and the Wasserman reaction. When

these observations give no conclusive evidence, a quantitative estimate of the sugar of the spinal fluid should be made.

The various examinations mentioned will, in the majority of instances, serve to differentiate epidemic encephalitis from other diseases clinically similar. Certain diseases, as will be emphasized in the following paragraphs, cannot be differentiated by all of these means.

1. MENINGITIS: (a) *Serous Type (Meningismus)*.—The cells and globulin in this disease show no deviation from the normal, but the pressure is always increased. These facts do not serve as a differential point for there are some cases of encephalitis in which the picture is the same.

(b) *Acute Purulent Meningitis*.—In the early stages, the marked polymorphonuclear increase is the most important differential point. In the later stages, the presence of cloudy opalescent fluid, with cellular increase of several thousand cells, predominantly of the polymorphonuclear type, present a picture which is never found in epidemic encephalitis. The quantitative sugar is usually increased or absent, and the colloidal gold solution may show a color change in the higher dilutions (right end of the curve). Bacteriologic examination may reveal organisms by smear or culture.

(c) *Tuberculous Meningitis*.—Examination of the spinal fluid, as thoroughly as has been suggested in the opening paragraphs, must be made in order to differentiate this disease serologically from epidemic encephalitis. It is emphasized in the literature that in tuberculous meningitis the cell count has a tendency to increase during the progress of the disease, while in epidemic encephalitis this is not the case. This observation, in our opinion, is of considerable importance, but we do not believe it to be invariably correct since there are cases of epidemic encephalitis in the course of which the cell count rises (Table 3). The picture of the spinal fluid in epidemic encephalitis may be identical with that of tuberculous meningitis with one important exception—the quantitative sugar. This is almost invariably diminished in the latter disease.

The colloidal gold curves seen in tuberculous meningitis are similar to those found in epidemic encephalitis, with the possible exception of paretic types of curves. No observations on the presence of these in tuberculous meningitis have been found. They are fairly common in cases of epidemic encephalitis.

(d) *Neurosyphilis and Syphilitic Meningitis*.—Epidemic encephalitis may be ruled out by a positive Wassermann reaction in the blood or spinal fluid, except in the rare coincidences of the two diseases. In such cases no differentiating point can be made. Cases in which the Wassermann reaction is not positive in the spinal fluid findings are also of no value when the history or clinical findings are clearly indicative of syphilis.

2. **POLIOMYELITIS:** This disease presents a picture which cannot be differentiated (at the present state of our knowledge) from epidemic encephalitis, in so far as the spinal fluid is concerned. Further researches on immunologic tests and quantitative sugar estimation on the spinal fluid may yield data of differential diagnostic importance.

3. **BRAIN TUMOR:** The presence of a high cell count is of extreme rarity in brain tumor; other than this there are no differential points in the diagnosis from epidemic encephalitis, in so far as the spinal fluid is concerned. In brain abscess, a mild pleocytosis, usually of polymorphonuclear cells, is often found.

4. **PARKINSONIAN AND ALLIED CONDITIONS:** Since the occurrence of the recent epidemic of encephalitis, this group must be divided into two parts: those caused by encephalitis and those not caused by this

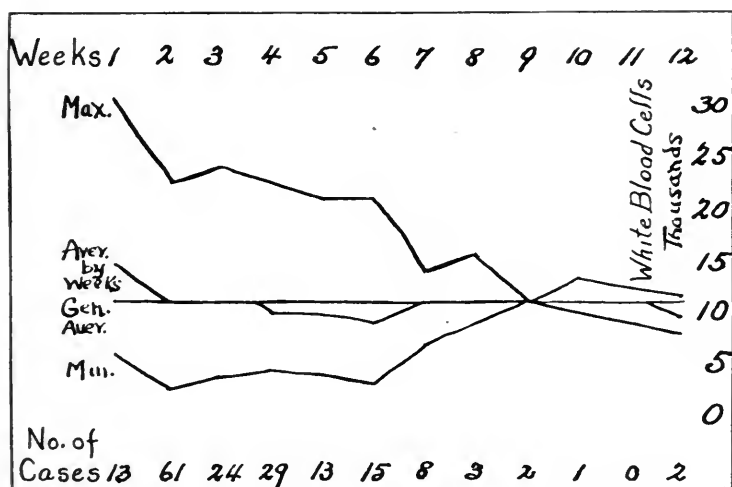


Chart 3.—Variation in leukocyte counts in the blood.

disease. In the acute and chronic stages of both of these groups, the spinal fluid findings are of great differential diagnostic importance. When the condition is due to encephalitis, several or all of the findings described in the foregoing may be found. When in this group a positive Wassermann reaction is found, the history of the case will decide the diagnosis rather than the spinal fluid findings, and the probability is that the case is due to syphilis. The parkinsonian syndrome may be on a syphilitic basis. When the condition is not due to encephalitis or syphilis, the spinal fluid is negative.

5. **MULTIPLE SCLEROSIS:** The differential diagnosis between this disease and epidemic encephalitis is, in our opinion, as far as the spinal fluid is concerned, not possible.

6. POLYNEURITIS: As there are no changes in the serology of the fluid in polyneuritis, while in most cases of epidemic encephalitis some change is found, this fact will serve as a differential point.

CONCLUSIONS

The spinal fluid findings in epidemic encephalitis have led us to certain general conclusions in regard to the nature of the disease.

The course of the disease, as illustrated by the serology of the spinal fluid, is extremely variable. A mild insidious or chronic type lasting for months exists, as is shown by the continuation of abnormal changes in the spinal fluid over long periods of time. An acute, fulminating type also exists and may or may not show serologic changes in the spinal fluid. Midway between these two extreme types of the disease is a combination of the acute, fulminating and chronic insidious types. In this type the spinal fluid findings indicate an active process which, after several weeks, subsides, leaving few or no serologic changes. Then the process lights up again with recrudescence of abnormal fluid findings, and may then continue to a fatal ending or what appears to be a recovery. It is striking that patients who have died have not, as a rule, shown any marked aggravation of the spinal fluid changes before death. The reason is that death occurs from an involvement of vital centers, which is not dependent upon a meningitis.

The low average of cell counts found in this disease indicates that the meninges are very little involved in the pathologic processes, the point of attack of the infection being predominatingly through the vascular and lymphatic systems. The meninges are not directly attacked.

The increase in the spinal fluid sugar, which has been noted whenever tests of this kind have been made, has appeared to us to be of great interest. This has been attributed to an involvement of the center of Claude Bernard in the floor of the fourth ventricle. However, it has also been shown by Aschner⁸ that "puncture of the floor of the third ventricle causes intense glycosuria." This arouses doubt in our minds as to the validity of the hypothesis that the hyperglycorrachia is necessarily due to a lesion of the fourth ventricle.

Furthermore, we have been impressed by the lack of correspondence between the amount of spinal fluid sugar, which is always increased, and the amount of blood sugar, which is never increased (Tables 4 and 5). This leads to an hypothesis which must be considered in

8. Aschner, B.: *Zur Physiologie des Zwischenhirns*, Wien. klin. Wchnschr. **25**:1042, 1912. Quoted from H. Higier: *Vegetative Neurology*, Nerv. & Ment. Dis., Monograph Series, No. 27, p. 35, 1919.

seeking an explanation for the quantitative sugar changes. The pathologic picture of epidemic encephalitis is essentially one of the blood vessels and the perivascular spaces. It seems possible that the thin membrane of cells, which normally retains within the blood a greater amount of sugar (0.80 per cent. to 0.120 per cent.) than that of the spinal fluid (0.040 per cent. to 0.060 per cent.) may, by being injured, permit the amount of sugar in the spinal fluid to approach that of the blood.

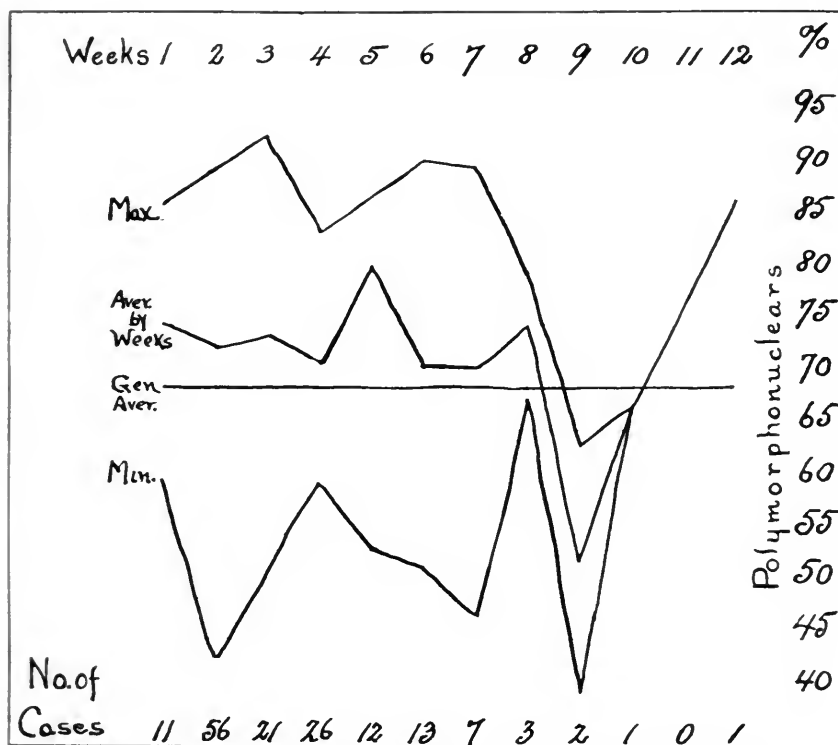


Chart 4.—Variation in numbers of polymorphonuclears in the blood.

The occurrence of rapid emaciation during the encephalitic process and of marked adiposity after it strongly suggests metabolic disturbances dependent on endocrine dysfunction. We believe that the observations on the relations of striate lesions to creatinism, the presence of cirrhosis of the liver in progressive lenticular degeneration (Wilson's syndrome), and of genital dystrophy, similar to Fröhlich's syndrome, occurring in midbrain lesions, are of great importance from both a theoretic and, perhaps, a clinical point of view. A hypothalamic-vegetative nervous system—endocrine connection, seems possible. We should like to urge an investigation of the basal metabolism as well as chemical

blood examinations in cases of epidemic encephalitis in the hope of obtaining more definite data on this very little considered subject.

The diagnostic value of the spinal fluid findings in epidemic encephalitis is mentioned in detail in the section on differential diagnosis. The combination of an increase of cells, globulin and sugar with changes in the colloidal gold curve constitutes a tetrad of laboratory findings of diagnostic importance. Findings other than these are not of diagnostic importance unless related to the clinical findings.

The blood picture gives evidence of a type of infection which does not call forth a polymorphonucleosis. It does not differ essentially from the picture shown by other similar toxemias and is mostly of importance in differentiating from organic diseases of nontoxic origin, such as brain tumor.

141 West Seventy-Fifth Street—74 West Forty-Eighth Street.

News and Comment

A FRENCH LEAGUE OF MENTAL HYGIENE

In April, 1920, the French Minister of Hygiene and Prevention instituted a Committee of Mental Hygiene. This has now been supplemented by an unofficial League of Mental Prophylaxis and Hygiene (*Ligue de prophylaxie et d'hygiene mentale*) which will be devoted to a study of all questions relating to mental health as it concerns the individual and communities. Indeed, the activities of the league as outlined will cover much wider ground than our own National Committee for Mental Hygiene. It proposes to open dispensaries for rational and harmless psychopaths, to influence legislation, to direct and assist research and to institute active propaganda among all classes.

The league desires affiliation and cooperation with similar organizations in other countries and will welcome inquiries and suggestions.

The president is Dr. Antheaume, a distinguished psychiatrist and publicist, and the secretary is Dr. Genil-Perrin, 99 Avenue de la Bourdonnais, Paris, France.

Abstracts from Current Literature

THE CHOROID PLEXUS IN ORGANIC DISEASES OF THE BRAIN AND IN SCHIZOPHRENIA. SADAMI KITABAYASHI, Schweiz. Arch. f. Neurol. u. Psychiat. 7:1, 1921.

The author introduces his subject by referring to the work done in investigating the glands of inner secretion in diseases of the brain, noting especially Mott's and his pupils' results in dementia praecox and the relations found between mental disturbances (apathy, weakness, dulness, etc.) and diseases of the pituitary and suprarenal glands. The choroid, on the other hand, has received little attention either through careful anatomic or clinical study so that the physiological importance and function of the choroid plexus is still little known, and it is only recently that some authors tend to consider it a body related to the glands of inner secretion.

The author's material was taken from three groups:

1. Choroid plexus from persons without psychosis dying from different diseases of the internal organs. In this group were included: (a) a child of 11 months, (b) a 10-year old boy (dying from perityphlitic abscess), (c) a 30-year old woman (tuberculosis), (d) a 35-year old man (lethargic encephalitis), and (e) a 55-year old man (cause of death, fracture and dislocation of hip with following pneumonia).

2. Choroid plexus from nonschizophrenic subjects suffering from chronic diffuse or focal lesions of the nervous system, in all eight cases falling into two subdivisions:

(a) Patients without delirium, among which were included one case each of general paralysis of the insane, chronic internal hydrocephalus and brain tumor (Cases 6, 7 and 8).

(b) Cases in which delirium was a prominent symptom, one each of senile dementia, arteriosclerotic dementia, and manic depressive insanity, idiocy with deafness and dumbness, and alcoholism with trauma (Cases 9, 10, 11, 12 and 13).

3. Plexus from schizophrenics. Eight cases were studied in which six patients died from somatic diseases (four cases influenza, one carcinoma ventriculi, one tuberculosis; Cases 14, 15, 16, 17, 18, 19, 20 and 21).

For purposes of study large blocks were taken from the fourth and lateral ventricles and to a lesser extent from the third ventricle, in each case removing a good sized portion of the ventricle wall so that the plexus could be studied in place and in connection with alterations in the adjacent tissues. Most of this material was embedded in celloidin and cut in serial sections of 15 m. Some paraffin sections were made when extreme thinness was desired.

The stains used were Nissl's toluidin blue, Van Giesson, hematoxylin.

The normal structure of the choroid plexus differs considerably in youth and later years. A typical normal plexus in childhood contains little connective tissue between the secreting cells. The cells are round and practically never vacuolated. No colloid masses, or chalky deposits or other foreign deposits are seen and no hyalin shrinking is seen at the top of the folia. Myeloid bodies are rarely seen. Little, if any, difference is observed in the plexus from the

different ventricles. In adult life (30 years in tuberculosis) slight changes are seen which are more marked in the man of 35 years in which some deeply staining bodies are seen; while in the presenile years (55-year old man) chalky concretions are seen, and the connective tissue is more or less atrophied or proliferated. The plasma and nuclei of the cells are almost normal. The ependyma and subependymal tissue is also almost normal.

GROUP 2: Severe organic disease of the brain with and without marked psychosis. In this group more or less marked alterations are seen in the choroid plexus in each case.

In Case 6—an agenetic brain with severe internal hydrocephalus—there is microplexia and atrophy of the folia cells (probably from pressure?), also many albuminoid bodies between the folia (which the author considers due to the illness from which the child died). The ependyma also shows both atrophy and proliferation, and there was proliferation of the subependymal tissue. The pericapillary connective tissue in accordance with the early age, was slight in amount.

The patient in Case 7 was a taboparalytic, who died in status epilepticus. The plexus showed no characteristic changes. The secreting cells were somewhat swollen and slightly vacuolated. There was also some exudate between the folia and on the ependyma, of a fibrinous character, but no formed bodies (Schollen). Such an exudate is seen frequently in patients with high fever, and is here attributed to the status epilepticus.

The patient in Case 8 had an angiosarcoma of the left gyrus fornicatus, corpus callosum, etc. The plexus showed much proliferation of the connective tissue in the perivascular and pericapillary spaces, hyaline degeneration and sclerosis of the secreting cells; between the folia red blood cells, lymphocytes and albuminoid bodies were seen.

The patient in Case 9 was a senile dement, 72 years of age, and showed changes of the plexus customary in advanced age: connective tissue proliferation and presence of chalky bodies, also severe degeneration of the secreting cells, which is not a part of the senile picture. The cells were swollen and had ameboid projections and were partly vacuolated, sclerosed or desquamated. The exudate between the folia also contained albuminoid bodies, and these bodies could be followed also into the subependymal tissue.

The manic-depressive patient in Case 10, 65 years of age, showed severe degeneration of the secreting cells, small nuclei, sclerosis of the cells, desquamation of whole rows of cells, homogenous degeneration of rows of cells and masses of concrement in the folia. Albuminoid bodies were also seen in the folia and could be followed into the subependymal tissues.

The 47-year old man in Case 11 suffered with chronic alcoholism, traumatic section of the spinal cord, hallucinations, ideas of persecution, unclearness and disorientation. Vascular changes were most notable in this case, the walls of both arterioles and capillaries showing hyaline degeneration; thrombi appeared in the capillaries. Extravasation of blood was seen around the arterioles. Many of the secreting cells showed vacuolization; some were atrophied, others sclerosed, swollen or desquamated.

An arteriosclerotic patient of 70 years, in Case 12, showed thickening, sclerosis, serpentine, narrowing and thrombosis of the arterioles with proliferation of the endothelia. The perivascular spaces were enormously enlarged and showed thick proliferation of the connective tissue. Single secreting cells or whole rows together were desquamated, and sclerosis and swelling of the

cells were seen. Albuminoid and amyloid bodies were present between the folia. The ependyma was seldom intact, and albuminoid bodies were seen in the subependymal spaces.

The patient in Case 13 was a deaf and dumb idiot, 53 years of age. The choroid plexus showed a medium grade of mass atrophy of the folia. The individual folia were mostly very small, sometimes sclerosed or swollen and often desquamated. Connective tissue proliferation was present and colloidal and chalky concretions. The subependymal tissue was rarefied, and concretions were also present in it.

GROUP 3: The choroid plexus in schizophrenic patients—Cases 14-21.

All the patients in this group showed almost the same clinical symptoms, only two of them not certainly showing hallucinations of hearing. Most of them were in middle life, only one being an aged person. In one case the disease had lasted three years, in four cases from ten to seventeen years, in one six years and in one twenty-one years. As complications influenza was present four times, tuberculosis, carcinoma and embolus of the lungs, each once. Although the ages varied from 25 to 60 years and the course and complications of the cases were different, the pathology of the choroid plexus was extremely alike in all of the cases: they all showed large masses of the folia rolled together, severe atrophy going on to sclerosis; sometimes the folia were attached to the ventricle walls or penetrated the adjoining brain substance (mostly in coincidence with amyloid bodies), frequently whole rows of folia were desquamated so that the mesodermal tissue lay bare and cysts were formed under the folia from shrinking of the connective tissues. A large number of glandular cells were irregular in size and form, many plainly atrophic, others sclerosed, great numbers vacuolated or swollen. The swollen cells not infrequently showed ameboid protuberances which projected into the ventricles. The nuclei of the secreting cells were frequently lessened in size, not perfectly round, now and then showed no histologic structure or had entirely disappeared; sometimes mitoses of the nuclei were seen. In many cases the arterioles of the folia were much thickened or sclerosed. The veins sometimes contained thrombi of blood plates and pigment clumps. In the pericapillary spaces of the folia atrophy, larger or smaller cysts and sometimes marked proliferation of the connective tissue, were seen. Chalky and other concretions were common. Hyaline degeneration of the tips of the folia was often present. In all cases cellular exudation was seen between the folia, mixed with larger and smaller amyloid or albuminoid bodies and old and recent extravasations of blood. Desquamated cells were also found between the folia, etc.

The ependyma frequently was lacking for considerable distances on the ventricle walls, and in such localities large numbers of rather large bodies staining pale in hematoxylin were seen in the subependymal tissue. Small cysts were also found in the subependymal tissues.

Some of these alterations were seen in Group 2 (hyaline degeneration of the tips of the folia, connective tissue proliferation) and even in the oldest number of Group 1, but in the schizophrenic patients they were more marked, more diffuse and more varied than in the patients of the other groups. Above all, rolling up of whole folia, their atrophy in mass or scleroses, were never absent.

From a consideration of the twenty-one cases, the author concludes that pathologic alterations of the choroid plexus can be differentiated into three groups:

1. Cases in which pathologic processes preeminently go out from, or are expressed in, the mesodermal elements of the plexus and in which change of form in the secreting cells (atrophy, increase or decrease of size) is more secondary in character.

2. Diseases of the choroid plexus in which the secreting cells are the point of attack of the pathologic process and in which the connective tissue elements of the folia are drawn into the diseased state sympathetically.

3. A combined form in which the pathologic process attacks both elements together or one following the other.

An example of the first or mesodermal type is seen in Group 2, Case 8, and also with some exceptions in Cases 11 and 12. The second form, parenchymatous, ectodermal, is exemplified in Cases 18 and 21 of Group 3, and the third form (the combined ectodermal and mesodermal) is seen in Cases 15 and 16, characterized by fibrinous and cellular exudates in the spaces between the folia, extensive atrophy of the secretory cells, thrombosis of the capillaries, etc.

After a somewhat lengthy consideration of the relationship of the pathologic findings to psychotic states, a consideration of the clinical character of amyloid bodies, etc., and a summary of the experimental and anatomic work on the plexus done by Monakow and his school and many others, the author draws the following conclusions:

1. Pathologic alterations in the choroid plexus, as commonly found in diseases of the brain and in psychoses, can be placed in three groups (1, 2 and 3, as shown in the foregoing).

2. Atrophic changes in the ependyma and subependymal tissue are specially marked in connection with Group 2 (acute and chronic changes originating in the glandular cells).

3. Where the changes described in pathologic Group 2 and the changes described above in the preceding paragraph are diffuse and coincident, particularly in the plexus of the inferior horn of the lateral ventricles and in the fourth ventricle, severe psychic symptoms are observed in life, particularly of a schizophrenic character (acute and chronic delirium, delusions and hallucinations).

4. The pathologic processes limited to the changes described in pathologic Group 1 may remain latent for a length of time, but if the processes go beyond a certain limit, it is probable that stupor, "dämmer" states, disorientation (sometimes with delirium) in time and space will result.

GURD, Ann Arbor, Mich.

ZUR LEHRE DER ERKRANKUNGEN DES STRIARENS SYSTEMS
(CONTRIBUTION TO THE STUDY OF DISEASES OF THE
STRIATE SYSTEM). CECILE and OSKAR VOGT, *J. f. Psychol. u Neurol.*
25:631, 1920.

Cecile Vogt in collaboration with Oskar Vogt, Bielschowsky, Brodmann and several other colleagues has for many years been devoting special attention to the anatomy, physiology and pathology of the striate system, and as far back as 1911 had formulated a striate syndrome. Several notable articles have been published by her since then, in particular one in this journal in 1918, volume 24, in collaboration with Oskar Vogt, in which the whole subject is

treated fully, and an attempt made not only to fix the striate syndrome, but also to relate the symptoms to various localities in the system and to explain their method of appearance.

The present article is a resumption and amplification of all the study done on the subject and forms a book of more than 200 octavo pages with nine illustrations in the text and seventy-eight full sized double tables at the end of the book.

The authors first give their reasons for their prolonged and intensive study of the striate system, namely, the relative ignorance both clinical and pathologic (at the time of the commencement of their studies) of the nervous system and its phenomena, and especially of the basal ganglions.

The striate system is then described in careful detail, first macroscopically and then microscopically. In the microscopic description much stress is laid on the fact that the caudate nucleus and the putamen are identical in structure, showing slight variations in the large and small nerve cells common to both, whereas the globus pallidus contains only one type of nerve cell, which is totally different from the cells of the other portions of the striate system.

Thirty-three cases of disease of the striate system are then described clinically and histopathologically, and a close analysis of the symptomatology and pathology is made with an attempt to explain the mechanism of the various phenomena.

Huntington's chorea receives special attention, five typical cases being analyzed. In general the pathology of the striate system is described as related especially to certain symptoms and not to diseases as a unit.

1. The striate system (in the narrow sense—caudate nucleus, putamen and globus pallidus) contains gray matter of physiologically higher and lower function. According to the destruction of gray matter of higher or lower physiologic moment, finer or coarser symptoms will appear. The loss of the finer functions of the striatum leads to continuous involuntary movements, while that of the coarser function of the globus pallidus causes rigidity with poverty of movements. In this way apparently unlike pictures of disease arise.

2. Their close connection is evidenced in that in the course of an individual disease the loss of the pallidus function is added to the loss of the striatum function, and may thus control the disease picture.

3. The intimate relationship of the picture of the disease (in the narrow sense) is made clear by the important fact that a one-sided affection of this neuron in any part results in a loss of striatum function on one side only.

4. The striate system represents a neuron system, disease of which in any part (in accordance with the principles laid down in 1) calls forth a related syndrome, while processes of disease in other parts do not bring about this syndrome. The striate system, therefore, affords us an example showing that for the understanding of clinical and physiologic phenomena the nervous system must be considered in, or divided into, neuron systems.

The striatum syndrome is made up of loss of function or of irritative symptoms. Loss of function is characterized by the following symptoms:

1. Striate akinesia, which is a component of poverty of mimic expression as well as of associated movements; changes of position; movements of orientation, protection or defense reflexes. This condition probably also expresses itself in a certain asthenia of the affected muscles.

2. Incoordination, which comes to view particularly in the bulbar musculature and in walking and standing.

3. Substrate (pallidus) hyperkinesis.

(a) Involuntary movements (choreatic movements, athetosis appearing only as a pseudo-Babinski sign, tremor; in great part exaggerated associated movements of expression, compulsory laughing and weeping) which (1) can always be voluntarily controlled for the moment, and (2) are called forth or exaggerated by peripheral stimulation and above all by psychic impressions (seelische Vorgänge) of an agreeable nature.

(b) Hypertonic conditions:

1. The duration and intensity of these are not yet sufficiently explained because we have not at our disposition pure striatum disease.

2. These conditions are strengthened by peripheral stimuli and above all by psychic stimuli, shown on finer analysis to be dependent on the emotions, but which are not exaggerated by continued stimulation and are lessened by nonirritative passive or active movements.

3. These conditions either attack by preference certain muscle groups or affect agonists and antagonists equally.

4. They cause a certain diminution of muscle strength or a slowness of movement.

5. Perhaps an occasional pathophysiological hypotonia as yet unclear.

6. The absence of other disturbances. Such a syndrome of disease will not be called forth by disease of any other portion of the cerebral gray matter so long as the striopetal and the striofugal paths are not involved. Disease of the striatum causes loss of function (Ausfalls erscheinungen) whose peculiarities are in proportional relation to its own individual architectonic.

The author's conception of a parallelism between architectonic and physiological differences is thus confirmed.

The following conclusions are drawn from the consideration of the striate syndrome as to the normal function of the striate system.

1. The striatum and pallidum of man show neither evolution nor devolution when compared with the same organs in the Cercopithecine brains. Therefore, the same function must be ascribed to these gray masses as that occurring in the lower apes.

2. The myelinization of the pallidus is so much earlier than that of the striatum and of the cortex that in all probability a time existed in ontogenetic development when pallidum reflexes uninfluenced by the cortex and the striatum represented the highest motor activity.

3. In the corticospinal tracts and in the cerebrum-pons-cerebellar tract a corticofugal path is furnished which, avoiding the pallidus and striatum, acts directly on the peripheral motor neuron.

4. The pallidal and striopetal path originates for the greater part at least in the thalamus and its contingent gray masses.

5. The subpallidary tracts do not act directly on the peripheral motor neuron.

In consideration of these anatomic facts the conclusion must be drawn from the pallidus syndrome (a general rigidity in limited tracts not seen in other pathologic conditions) that the pallidus gray matter exerts an inhibitory action normally on subpallidal gray centers.

From the striatum syndrome is to be predicated that the pallidus is the center for numberless primitive kineses.

The authors close their work with the expression that further intensive histopathologic and clinical study of the striate system will amply reward the worker both by increased knowledge of the diseases of the system and also of a number of general anatomic and physiologic questions.

PATIENTS WITH MENTAL DISEASE, MENTAL DEFECT, EPILEPSY, ALCOHOLISM AND DRUG ADDICTION IN INSTITUTIONS IN THE UNITED STATES, JAN. 1, 1920. HORATIO M. POLLOCK, Statistician, State Hospital Commission, and EDITH M. FURBUSH, Statistician, The National Committee for Mental Hygiene, *Ment. Hygiene* 5:139 (Jan.) 1921.

Data for this comprehensive study were obtained from practically all state, city, county and private institutions caring for the classes enumerated, and from thirty public health service hospitals. The institutions are classified thus:

1. Number of institutions.....	625
a. Public	388
b. Private	237
2. Public institutions for mental diseases:	
a. State hospitals	156
b. Federal hospitals	2
c. County and city institutions (not including those for temporary care).....	125
d. Institutions for temporary care.....	21
3. State institutions for mental defectives.....	32
4. State institutions for mental defectives and epileptics..	11
5. State institutions for epileptics.....	10
6. City institutions for mental defectives.....	1
7. Private institutions:	
(a) Having mentally diseased patients only.....	60
(b) Having mental defectives only.....	27
(c) Having epileptics only	6
(d) Having mental defectives and epileptics only....	19
(e) Having inebriates (alcoholics and drug addicts only)	12
(f) Having more than one of these classes (excluding "d" above)	113
8. Public Health Service hospitals.....	30

Patients with Mental Disease.—On Jan. 1, 1920, there were 232,680 patients with mental disease actually in institutions in the United States. Of these, 200,109 were in state hospitals, 21,584 in county or city institutions, 1,040 in institutions for temporary care, 709 in Public Health Service hospitals and 9,238 in private hospitals. In 1918, the total number of patients with mental disease reported on the books of the several institutions was 239,820. Of this number 15,863 were reported as on parole from the state hospitals on the day of enumeration. The number on parole from private institutions was not obtained, but it is believed to be small. The number of patients actually in the institutions on Jan. 1, 1918, was approximately 223,957, or 8,723 less than the number on Jan. 1, 1920.

Of the 232,680 patients with mental disease in institutions on Jan. 1, 1920, 121,031 were males and 111,649 females. The percentages were 52 and 48, respectively. The number of males to each 100 females was 108.4. In 1918, the number was 110.6; in 1910, 110.8; in 1904, 109.6; in 1890, 107.4, and in 1880, 101.6.

With the marked decline in alcoholic psychoses and the gradual reduction of the syphilitic psychoses, it is probable that the excess of males in the hospitals for mental diseases and among admissions to them will ultimately disappear.

Mental Defectives.—The total number of mental defectives in institutions in the United States on Jan. 1, 1920, was 40,519. Of these, 34,836 were in state institutions, 2,732 in other public institutions and 2,951 in private institutions.

Mental defectives were reported in state institutions in all states except Delaware, Georgia and New Mexico, although on Jan. 1, 1920, there were fourteen states that had no separate institutions caring for such patients. The mental defectives reported in state institutions in Alabama, Arizona, Arkansas, Florida, Louisiana, Mississippi, Nevada, South Carolina, Tennessee, Utah and West Virginia were cared for in hospitals for mental disease. Of the 34,836 mental defectives in state institutions, 28,833, or 82.8 per cent., were in state institutions especially established for their care.

Compared with the census of mental defectives of Jan. 1, 1918, there has been an increase of 2,047 in state institutions, a decrease of 756 in other public institutions and a decrease of 153 in private institutions. The increase in total patients amounts to 1,138. As only a small portion of the total number of mental defectives is cared for in institutions, the census throws no light on the prevalence of mental defect in the general population.

Epileptics.—Exclusive of the epileptics included among the patients with mental disease, there were on Jan. 1, 1920, 14,937 epileptics under treatment in institutions of the United States. Of these, 13,223 were cared for in state institutions, 859 in other public institutions and 855 in private institutions. Compared with the census of Jan. 1, 1918, there was an increase of 2,781 epileptics in state institutions, a decrease of 92 in other public institutions and an increase of 304 in private institutions. The total increase in all institutions was 2,993. The prevalence of epilepsy in the general population is not indicated by these figures as only a small proportion of the total number of epileptics is in institutions.

Colorado, Delaware, Georgia, Nebraska, New Mexico and Washington report no epileptics in state institutions other than those included among the patients with mental disease. In only twelve states are epileptics cared for in city or county institutions. Nearly one half of the epileptics in private institutions are reported from Pennsylvania.

Alcoholics.—The alcoholics in institutions in the several states on Jan. 1, 1920, other than those included among the patients with mental disease, numbered 1,163. Of these, 587 were cared for in state institutions, 331 in other public institutions and 245 in private institutions.

In the census of 1918 the alcoholic patients and drug addicts were included in one group under the term "inebriates." In the present census the two classes were separated. In view of the oft-expressed opinion that restriction of the sale of alcoholic beverages would greatly increase the use of drugs, it is noteworthy that the total number of alcoholic and drug inebriates in institutions in the United States decreased from 3,565 on Jan. 1, 1918, to 1,971 on Jan. 1, 1920. On Jan. 1, 1917, the total inebriates in institutions in this country numbered 4,891, or two and one-half times the number shown by the 1920 census.

While there are many alcoholic patients and drug addicts outside of institutions, the marked reduction of the number in institutions indicates that the number outside is also decreasing. This conclusion is further supported by the fact that the number of alcoholic and drug patients among first admissions to the New York State hospitals for mental diseases has markedly declined in recent years.

Drug Addicts.—The drug addicts in institutions in the United States on Jan. 1, 1920, numbered 808. Of these, 314 were in state institutions, 78 in other public institutions and 416 in private institutions. These figures throw little

light on the prevalence of drug addiction as most drug addicts are not receiving institutional care. As no other separate census of drug addicts in institutions has been taken in recent years, no basis of comparison is available.

Of the 808 drug addicts in institutions, 544 were males and 264 females, or two males to each female. New York is the only state in which the number of drug addicts reported reached 100.

Increase of Patients with Mental Disease.—The institutional care of the insane has increased enormously since 1880, when the first separate federal census of the insane in institutions was taken. The absolute numbers of patients under treatment, as well as the rates of patients to population, have increased at each succeeding census.

PATIENTS WITH MENTAL DISEASE IN INSTITUTIONS, 1880-1920

Year	Number	Per 100,000 of General Population
1880.....	40,942	81.6
1890.....	74,028	118.2
1904.....	150,151	183.6
1910.....	187,791	204.2
1918.....	223,957 *	217.5
1920.....	232,680 *	220.1

* Excluding paroles.

BAILEY, New York.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF CARBON MON-
OXID POISONING. R. M. STEWART, J. Neurol. & Psychopath. 1:2
(August) 1920.

Two views are expressed concerning the effects of carbon monoxid on the system: one holds that the gas produces an anoxemia by combining with the hemoglobin and preventing proper oxygenation of the blood, while the other holds that the gas has a specific action on the tissues of the nervous system. The author points out that the dyspnea so characteristic of asphyxiation is lacking in gas poisoning and infers that the respiratory center, with other centers, is interfered with to such an extent that it cannot respond to the increased demand for oxygen.

The characteristic symptoms of carbon monoxid poisoning are: headache, vomiting, impairment of higher mental functions, impairment of the special senses and voluntary motor power—and in severe cases loss of consciousness and death. In the discussion of the pathology, Mott is quoted as having found capillary hemorrhages and various chromatolytic changes in the brain cells with widespread fatty degeneration of the endothelium of the blood vessels. Davies is reported as unable to find punctate hemorrhages in his case. In those who survive, it is found that the symptoms predominating are referable to the nervous system, leading to the conclusion that here is found the main pathology.

The author reports a case coming under his observation and in which death occurred on the twenty-fourth day, and gives the gross and histologic findings. He states that death occurring early fails to give the pathology that is found in those cases that continue for some time. Clinically, his patient presented a picture of extreme debility, constipation, cyanosis, normal temperature, albuminuria, unequal pupils, exaggerated knee reflexes, wasting of the small muscles of the left hand, moderate stupor, apathy, and difficulty in being persuaded to take food. Four days later (seventh day of illness), his temperature rose to 100 F., his torpor gave way to a condition of restlessness, and he frequently

cried out. Five days later a spinal fluid examination revealed a lymphocytosis and positive Noguchi reaction. Two days later he seemed better and tried to speak, but showed incontinence and marked wasting of the muscles of the thenar and hypothenar muscles of the left hand. At the end of the next four days wasting was present in the muscles of the other hand, and on the following day he died.

At necropsy the gross findings were: hyperemia of the pia with thickening evenly distributed; a grayish white line near the surface in the gray matter running practically throughout the entire cortex suggesting cerebral softening. There were no punctate hemorrhages. The globus pallidus had a small area of softening bilaterally in exactly similar situations and similar in size.

Histologically, there was a zone of softening in the deeper layers of the cortex, which was present in all sections. Above and below this line of softening the cells were markedly altered but still recognizable, whereas in the line they had completely disappeared or nearly so. In the pia arachnoid there was evident congestion of the blood vessels, with numerous hyaline thrombi. The cellular proliferation in the pia consisted of plasma cells, fibroblasts, polyblasts and red cells. In the line of softening there was a large amount of disintegrating lipid substance, which stained deeply with acid dyes. All traces of axis cylinder were lost in this area, but observed well, above and below. The neuroglia tissue showed active proliferation. There was increased vascularity with formation of new vessels and the presence of many hyaline thrombi. Parenchymatous and interstitial changes were found in the cortex not immediately involved in the softening process. The white matter showed widespread degeneration of the myelin sheaths, with various morbid changes in the axis cylinders, increase in neuroglia, and hyaline thrombi in vessels, but no free hemorrhages. In the basal structures the globus pallidus showed changes confirmatory of the gross picture, with a large number of Kornchenzellen filling it. The optic thalamus showed hyalin thrombosis. The cranial nerves showed myelin degeneration more particularly marked in the vagus than in any other locality. The cerebellum showed colloid substance beneath the pia, hyaline thrombosis, chromatolytic changes in the Purkinje cells, and myelin degeneration. The pons, medulla and cord showed the same myelin and axis cylinder changes, with practically no normal cells in any region. Ganglion cells were similarly affected. Reich corpuscles were found in the cord lying free in the tissue spaces, apparently having no relation to blood vessels, and arranged in rows (on longitudinal section) as though they followed a degenerated axis cylinder.

The author, in his remarks, interprets the widespread parenchymatous degeneration on the basis of an hematogenous intoxication, with the vascular arrangement in the cortex accounting for the peculiar distribution of the degeneration. Experimental observations have disclosed the fact that the infragranular layers of the cortex are the anastomotic points of the long and short branches of the pial vessels, and hence there is found here the richest blood supply. The necrosis in this area cannot be explained by mere deprivation of oxygen (despite the greater vascularity) for experimental work has shown that the effect of deprivation is more generally distributed. The carbon monoxid therefore acting as an exogenous poison, and being present here in larger quantities because of the fine mesh of capillary formation, attacks with greater virulence, and produces the widespread but selected area of necrosis. Of course, other factors must be considered also, such as arrest of circulation favored by the anastomotic arrangement as mentioned, by the fine caliber of the capil-

laries and by the hyaline thrombi which were found everywhere in the cortex. Softening in the striate body is apparently characteristic of gas poisoning but the explanation for it is perhaps more difficult on the basis of vascular arrangement than in the cortex. The distribution of blood vessels evidently has little to do with it since areas supplied by the same vessel are not similarly affected. However, it was noted that the most marked degenerative change was around the blood vessels. This, however, is not sufficient to explain the completeness of the softening of the globus pallidus and its restriction to this area. There remains, then, the theory that the gas has a selective affinity for the tissues of the lenticular nucleus.

The presence of plasma cells, lymphocytes and red cells, the increased vascularity of the cortex and other changes are accounted for on the basis of some preexisting condition, possibly a syphilitic infection. The final conclusion drawn is that the carbon monoxid acts indirectly on the nervous tissue by diminishing the supply of oxygen and directly by specific action on the tissues themselves.

PATTEN, Philadelphia.

ZUR FRAGE DES "PARKINSONISMUS" ALS FOLGEZUSTAND DER ENCEPHALITIS LETHARGICA (REGARDING PARKINSONISM AS A SEQUEL TO LETHARGIC ENCEPHALITIS). ROBERT BING, Schweiz. med. Wchnschr. 1:4 (Jan. 6) 1921.

Since the first epidemic of lethargic encephalitis in Vienna, in 1916, numerous types of this protein disease have been encountered. The 1920 epidemic in Austria presented a preponderance of a tabetic type, characterized by absence of patellar reflexes and pupillary disturbances. True Argyll Robertson phenomena were noted, which, as the author states, had up to this time been considered as distinctly syphilitic in origin. In recent epidemics, cases have presented themselves showing a marked similarity to Parkinson's disease.

In the majority of cases of postencephalitic parkinsonism, a distinct resemblance to paralysis agitans may be noted. Some cases are symptomatically identical "so that without an anamnesis, the diagnosis would, without doubt, be Parkinson's disease.

The writer presents two cases, the first having a history of three years' duration, being typically parkinsonian in type, and showing no indications of improvement. The second case, with a history of one year, was not so characteristic of paralysis agitans and recovery was fair. The first case was that of a man, aged 47, whose trouble began in December, 1917, with lethargy, a slight temperature, bilateral ptosis, anisocoria, nystagmus and tremors. By February, 1918, the patient was up and about but during that summer a gradual and general muscular rigidity appeared, since which time he has developed a mask-like facies, a typical posture and a "démarche a petits pas," a pill-rolling tremor, etc. This case Bing feels is a typical paralysis agitans with the usual prognosis.

The second case, a man of 46, in February, 1920, had a slight temperature with loss of pupillary reflexes, diplopia and tremor. Later there were muscular weakness with increasing slowness of motion, slight paresis of the left arm and left side of the face, and some difficulty in swallowing. Gradually the patient improved and resumed his work though at the end of a year there still remained some muscular weakness with salivation. Bing considers this an

atypical case, not distinctly parkinsonian, and a better prognosis might be offered.

The author does not believe that the tremor lends itself to prognostic value, and he cannot agree with the work of Sicard and Paraf in which they recognize three types, depending on the degree of tremor on which they base their prognosis. Even though the author's own cases fit in their classification, a prognosis based on tremor is rash, for not infrequently the cases "sine agitatione" become rapidly and progressively worse. This also has been noted by Babinski, who observed that the pill-rolling type may recover. Bing believes, and is in accord with Netter, that the postencephalitic Parkinson's disease and the usual paralysis agitans are the same. It is the author's belief that just as sporadic cases of poliomyelitis may occur, so sporadic cases of paralysis agitans may occur. He further believes that Parkinson's syndrome may occur on the basis of various other conditions, such as status lacunaris cerebri, Bechterew's hemitonia postapoplectica, striate syndrome of Cecil Vogt, etc., the outstanding feature in these cases being the localization of the lesions, and the site being in the corpus striatum, the hypothalamus and midbrain regions. Bing believes that all the histopathologic work, as done by Jelgersma in relation to the striothalamic fibers and ansa lenticularis, that of Ramsay Hunt with the globus pallidus, Trétiakoff with the substantia nigra, point in one direction, namely, that they indicate a lesion of the striate body and its neighboring parts.

The tremor of paralysis agitans, according to the work of Economo and Karplus, may be attributed to the nucleus ruber, as was demonstrated by their experimental work and also by the work of Wilson and Holmes. The extrapyramidal rigidity (so-called because of absence of the Babinski and similar reflexes) is considered by Wilson to be due to an abolition of the functioning inhibition existing in the striate via thalamus and pyramidal cells. According to Lhermitte, the globus pallidus has special relation to tonus. Bing states that the secretory disturbances, such as salivation and hyperhidrosis, are due to 'tween brain disturbances, for he states that we cannot doubt the importance of considering vegetative tracts passing through this region. These secretory disturbances are frequently found in postencephalitic parkinsonian cases. Netter attempted to show that this salivation was due to infection of the salivary glands and not to a central manifestation. This, however, is not in accord with Bing's view.

The writer does not feel that the postencephalitic parkinsonian cases are due to the presence of the virus, but that the initial lesion is so intense as to cause a disease picture which is distinctly progressive and incurable.

Trétiakoff and Bremer at necropsy examination in a postencephalitic Parkinson's case found some inflammatory residuals, also bilateral degenerative atrophy of the substantia nigra. This concurred with Bing's view that a definite pathology is established and that the remaining syndrome is not due to the presence of the virus.

The writer feels that the present epidemic encephalitis has presented such a complicated and vast array of lesions in the 'tween brain and midbrain that a systematic histopathologic study of this rich material should certainly, in conjunction with clinical findings, solve many of the puzzles of brain localization.

MOERSCH, Rochester.

DECLINE OF ALCOHOL AND DRUGS AS CAUSES OF MENTAL DISEASE. HORATIO M. POLLOCK, Statistician, State Hospital Commission, Ment. Hygiene 5:123 (Jan.) 1921.

The data which form the basis of this article were compiled from the statistical cards of first admissions received by the New York State Hospital Commission from the thirteen civil state hospitals under its jurisdiction during the twelve fiscal years beginning Oct. 1, 1909, and ending June 30, 1920.

The total first admissions of alcoholic patients during the twelve fiscal years were 5,317 of which 4,007 were males and 1,310 were females. During the five fiscal years 1909 to 1913, inclusive, the annual number of alcoholic cases averaged 574 and varied but little. In 1914, there was a marked drop in the number, and this was followed by another drop in 1915. In 1917, a marked increase occurred but this was followed by a rapid decline until, in 1920, the total alcoholic first admissions numbered only 122. The percentage of alcoholic cases among first admissions dropped from 10.8 in 1909 to 1.9 in 1920.

RATE OF ALCOHOLIC FIRST ADMISSIONS TO THE CIVIL STATE HOSPITALS FOR THE INSANE PER 100,000 OF THE GENERAL POPULATION OF THE STATE, 1909-1920

Year	Number	Rate Per 100,000 General Population
1909.....	561	6.3
1910.....	583	6.4
1911.....	591	6.4
1912.....	565	6.0
1913.....	572	6.0
1914.....	464	4.8
1915.....	345	3.6
1916.....	297	4.0 *
1917.....	594	6.0
1918.....	354	3.5
1919.....	269	2.6
1920.....	122	1.2

* Reduced to yearly basis.

It will be noted that the rate of alcoholic first admissions per 100,000 of the general population declined from 6.4 in 1910 to 1.2 in 1920.

Intemperate Use of Alcohol.—If the facts concerning the decrease in alcoholic insanity stood alone they might be interpreted as being due to changes in diagnosis rather than to changes in the use or influence of alcohol. Additional light is thrown on the matter by the record of the intemperate use of alcohol by first admissions prior to the onset of the mental disease.

It appears that of the first admissions of 1909, 44.2 per cent. of the males and 15.1 per cent. of the females were intemperate users of alcohol. In 1920, only 20.3 per cent. of the males and 3.7 per cent. of the females were reported in the intemperate group.

In considering these facts in connection with the prohibition amendment, it should be remembered that the amendment was in force for only five and one-half months of the fiscal year that ended on June 30, 1920. Of the 122 new cases of alcoholic insanity admitted to the civil state hospitals during the year, seventy-five reached the hospitals before Jan. 16, 1920, and forty-seven after that date. As nearly all forms of alcoholic insanity result from long continued and excessive use of alcohol, it would be expected that some cases

would develop after the public sale of intoxicating liquors ceased. The great reduction in the rate of admissions of new alcoholic cases since the amendment went into effect indicates that excessive drinking has been much lessened, although not entirely stopped.

In this connection it should be remembered that for several years prior to the passage of the prohibition amendment there had been a gradual decline in excessive drinking and that during the greater part of the war traffic in distilled liquors was forbidden.

Mental Disease Due to Drugs.—It was feared by many that the discontinuance of the public sale of alcohol as a beverage would result in increased indulgence in the use of narcotic drugs. It was found, however, that the drug cases among first admissions have declined rather than increased during the past year.

Drugs have never been prominent among the causes of insanity in New York State. The highest number of drug first admissions to the civil state hospitals recorded in any one of the past twelve years was thirty-six in 1914. These constituted about 0.6 per cent. of the total first admissions. Since 1914, the annual number of drug cases has declined, there being but eleven, or less than 0.2 per cent. of all first admissions in 1920.

BAILEY, New York.

DREI FAELLE VON FAMILIAERER ZEREBRALER KINDER-LAEHMUNG (THREE CASES OF HEREDITARY INFANTILE CEREBRAL PALSY). KRETSCHMER, *Deutsch. med. Wchnschr.* **46**:1241 (Nov. 4) 1920.

The author presents the history of three brothers in whom, during the twelfth year of life, a slowly progressive disease made its appearance. The disease was characterized by a spastic paraplegia, or tetraplegia, appearance of club foot, partial optic atrophy, impediment of speech and a more or less marked mental retardation. All three brothers were physically well until the onset of the trouble. The two younger brothers did poorly in school from the start, while the older showed but little mental change even after the onset of his affliction. The rate of progress up to the time of the author's examination was practically the same in all three cases.

The family history was negative throughout. A younger sister, aged 15, was well, both mentally and physically. The author gives the complete findings in the three cases.

CASE 1.—F. M., aged 20, up to the age of 12 progressed rapidly, at no time presenting any abnormality. At 12 years of age, stiffness appeared in the lower limbs, and his progress in school became retarded due to slowness in his speech. An examination made in November, 1919, showed that he was well built and in good health. He showed a general spasticity, most marked in the lower limbs, with inability to walk unless aided. He walked with a scissors gait. Motor power was impaired only by rigidity. No ataxia was noted in the various tests. Bilateral pes equino-valgus was present. The deep reflexes were exaggerated and the Babinski sign was present. Speech was slow and stammering. Memory was poor for recent events, with impairment in general knowledge. The fundi showed a temporal pallor of the optic nerve heads. Examination was otherwise negative. There was no nystagmus, sensation was intact, etc.

The two brothers, aged 16 and 15 years, respectively, presented practically the same findings. Both were well up to the age of 12. They were never very

bright and had marked difficulty in learning. At the age of 12 rigidity appeared in the lower limbs. This general condition gradually progressed until the patients presented practically the same picture as the older brother. At no time was ataxia noted. The one (aged 16) presented lost patellar and Achilles' reflexes. The other (aged 15) had diminished abdominal reflexes.

In the differential diagnosis, Kretschmer first takes up Friedreich's ataxia, which he states may at times closely resemble hereditary cerebral palsy. The absence of ataxia in all three cases is, however, sufficient to exclude this condition.

The familial occurrence of multiple sclerosis (Oppenheim) is next considered. In the author's cases the absence of nystagmus, intention tremor and the presence of mental enfeeblement from onset, speak against such a diagnosis.

The author believes that his cases conform most closely to Strümpell's hereditary and familial form of spastic spinal paralysis, which by many is considered a form of hereditary cerebral palsy. To tell to which form of infantile cerebral palsy these cases belong is difficult. The author believes that the so-called Little's disease may be ruled out because of the late development, even though Little's disease may be familial. Gower's abiotrophy, which has been substantiated by Schaffer, Vogt, and others, in cases of this type in which marked ganglion cell changes were found, seems to the author to explain the condition presented. The majority of these ganglion cell changes were found in cases of familial amaurotic idiocy. In the Vogt-Spielmeyer type of this disease, the onset occurs late; in patients from 4 to 16 years of age, the progress is slower and the disease has the same symptoms, but in place of a macular change, it shows a simple optic atrophy. The Vogt-Spielmeyer type is not racial, but alcoholism in the parents seems to play a part. Kretschmer believes that his cases would perhaps fit best into some such group, even though his patients did not show an impairment of vision.

Even though the immediate family history is negative for nervous afflictions, the fact that the three males were affected and not the female, is indicative of an abiotrophy. Other hereditary anomalies show this tendency to sex differentiation, such as hemophilia, colorblindness, etc.

The question of a hereditary syphilis must be considered as it may play a part in Little's disease. The repeated negative Wassermann reactions, the late appearance of the symptoms, the absence of miscarriages and a healthy daughter who has already passed the period of onset by two years, argues against such a possibility.

The author in closing brings up the subject of inheritance, again calling attention to the possibility of transmission of the disease as in hemophilia, colorblindness, etc. He believes that necessarily similar conditions have occurred in the progenitors, or it would not be possible to explain the appearance of the disease in the three brothers at the same age. The prognosis in these cases is hopeless, and as a rule these patients spend the remaining years of their life in public institutions.

MOERSCH, Rochester, Minn.

ETUDE EXPERIMENTALE DES LESIONS COMMOTIONNELLES DE LA MOELLE EPINIERE (EXPERIMENTAL STUDIES OF CONCUSSION OF THE SPINAL CORD). G. ROUSSY; J. L'HERMITTE, and L. CORNIL, *Ann. de méd.* 8:355 (Nov.) 1920.

The authors report the effects of concussion from direct and from indirect spinal traumatism.

The first type of injury was inflicted on experimental animals (rabbits and guinea-pigs) by direct blows of a trepanning hammer on the spinal column.

The strength of the blow is well indicated by the statement that in several animals rupture of either a viscera or an important blood vessel occurred with rapid death. Among sixteen animals subjected to concussion in this fashion only seven were retained as suitable for further research.

These seven animals were then killed at varying dates after traumatism: one after three and one-half months, others after twenty-one, twelve and eight days, and three immediately following the trauma.

Indirect traumatism was used in one instance. The animal (dog) anesthetized was fixed solidly with his back along a plank. Then a dozen blows were struck on the plank so violently that the spine was severely jarred. The animal showed no effects until two months later. At that time, a cachexia began which progressed to death six months after the traumatism.

The following tissue alterations were found following the direct trauma (even present in the animals killed immediately after the trauma): an acute degeneration of the myelinic fibers, chiefly in the anterolateral tracts and posterior columns; small areas of necrosis involving the neuroglia and neurons; intense coloration of the nerve fibers of the radicular zones with the presence of vacuoles; and dilatation of the ependymal canal. The nerve fiber degeneration was shown by hypertrophy of the axis cylinders, which had become moniliform, owing to fragmentation. The writers especially emphasized the minimal character of the changes in the cells of the gray matter. In no case did they encounter hemorrhage as part of the lesion.

The concussion of the spine by indirect traumatism gave a different histologic picture as the damage was limited to the nerve cells of the anterior horns and only appeared microscopically: atrophy of the cell body, granulation of the cytoplasm, diminution of the Nissl bodies, and regressive alterations of the nucleus. The writers are inclined to consider that their findings support the thesis of Kirchgässer that the intensity of nerve cell alterations is in inverse ratio to the violence of the concussion.

In the summary the authors emphasize anew that concussion brought about by direct traumatism affects the myelinated fibers of the spinal tracts while that from indirect traumatism exercises a less intense action on the cells of the gray matter.

These findings are confirmatory of the work of some of the former investigators, particularly of the observations of Schmaus and of Jakob.

DAVIS, New York.

UEBER REZIDIVIERENDE SCHLAFTE LAEHMUNGEN NACH
FRUEHERER EPIDEMISCHER POLIOMYELITIS (RECURRING
FLACCID PARALYSIS FOLLOWING EPIDEMIC POLIOMYELITIS).
GEORGE BALLER, *Neurol. Centralbl.* **39**:658, 1920.

The patient, a girl aged 14, had poliomyelitis in 1909, which affected the right arm and leg. The leg recovered completely in fourteen days, the arm in six weeks with slight, but permanent, weakness and atrophy. In the spring of 1917, a few days after she had had diphtheria, she gradually developed a complete flaccid paralysis of both legs. This was associated with severe pains and reduction of sensibility. She recovered in six weeks. Three months later following a cold with fever, and pains in the legs, a flaccid paralysis in the lower extremities recurred with recovery in three weeks. In the fall of 1917 and again a year later, like recurrences of paralysis with fever and pains occurred, from which she recovered in three and five weeks. In February,

1919, she again had severe tonsillitis. Five weeks later pains developed in the right arm and within two days a complete flaccid paralysis occurred. This had been present six weeks when the author's observation began.

The child was pale, weak, not "nervous," without fever and with sound organs. The tonsils were enlarged without plugs or exudate. Tests were negative for malaria and diphtheria bacilli. The Wassermann test of the blood was also negative.

The neurologic examination was negative except in the right arm where were noted: complete flaccid paralysis except for slight flexion and extension of the first phalanges of the fingers and slight atrophy of the arm and forearm. The arm fell in proportion to its weight; the skin of the hand was slightly cyanotic; triceps, periosteal and direct muscular irritability were greater on the right than on the left; sensation was normal; and reaction of degeneration was absent.

Definite improvement was first noted seven weeks later in the flexion and extension of the fingers and weak flexion of the elbow. Gradually, improvement extended proximately with complete recovery in six months from the onset with a residual atrophy no greater than followed the illness of 1909. The illness of 1909 was observed in the Marburg Clinic and diagnosed as anterior poliomyelitis.

The author recognizes the difficulty of accurate diagnosis and considers hysteria, paroxysmal paralysis, malaria, myasthenia, multiple sclerosis, diphtheritic neuritis, idiopathic or rheumatic polyneuritis, believing that these can be excluded with reasonable certainty. The preservation of the tendon reflexes is unusual, but he suggests that some associated changes in the pyramidal tracts may explain this. The specific agent here at work resembles that of anterior poliomyelitis so closely that this tentative diagnosis is offered.

SHELDEN, Rochester, Minn.

DIE AFFEKTIONEN DES NERVENSYSTEMS DURCH AKUTE
INFEKTIONSKRANKHEITEN SPEZIELL DIE GRIPPE (AFFEC-
TIONS OF THE NERVOUS SYSTEM RESULTING FROM ACUTE
INFECTIOUS DISEASES, ESPECIALLY FROM INFLUENZA).
HAROLD SIEBERT, Monatschr. f. Psychiat. u. Neurol. **48**:149 (Sept.) 1920.

The author groups his cases in four classes:

1. Pure psychotic disturbances.
2. Psychotic manifestations associated with meningitis or encephalitic processes.
3. General neuroses.
4. Peripheral nerve lesions.

1. Pure Psychotic Disturbances: These may occur either at the onset of the disease or after defervescence. In the author's experience all those occurring at the onset of the disease were accompanied by meningitis or encephalitic manifestations. Psychoses uncomplicated by evidence of cerebral disease occurred during or following defervescence. Some were typical amentias, others showed a katatonic syndrome, still others a manic picture. In all these the author stresses the exogenic factor; he believes the influenza toxins can produce any of these pictures, quite independent of endogenic factors. In general, the outlook is good for a complete resolution of the mental symptoms. Where an endogenic basis does exist, the outlook is not so good.

2. Psychotic manifestations associated with meningitic or encephalitic processes.

3. General Neurosis: There can be no doubt about the development of neurasthenic symptoms as a result of infectious processes, and we need not assume any endogenic basis. However, in many cases both factors play a rôle, and the extent of the endogenic basis probably has much weight in determining the ultimate outlook. In the pure exogenic neuresthenias there was a striking muscular weakness and muscle tenderness; on the affective side, fear was a frequent symptom; sleeplessness and headaches were common. On the vegetative side, there was a tendency to vasomotor disturbances, palpitation, increased secretion and excessive sweating.

4. Affection of Peripheral Nerves: This occurred in the form of neuritis and polyneuritis.

SELLING, Portland, Ore.

PARAPLEGIE EN FLEXION D'ORIGINE CEREBRALE PAR NECROSE SOUS-EPENDYMAIRE PROGRESSIVE (FLEXION PARAPLEGIA OF CEREBRAL ORIGIN AS A RESULT OF PROGRESSIVE SUB-EPENDYMAL NECROSIS). P. MARIE and C. FOIX, *Rev. Neurol.* **27**:1 (Jan.) 1920.

Marie and Foix report a case of flexion paraplegia (in a woman, aged 70) in which there was determined at necropsy, extensive subependymal necrosis of both paracentral lobules, resulting apparently, from partial obliterations in the course of the anterior cerebral arteries.

There was a history of paraplegia of many years' duration, which at the time of examination, was definitely of the flexion type, although the authors are unable to state whether or not this had always been the case. Locomotion was impossible. The characteristic attitude was that of hyperflexion of the legs on the pelvis, associated with marked movement limitation of the knees and hips, although passive ankle movement was still possible. The lower extremities showed marked muscular weakness with atrophy, particularly of the quadriceps femoris. Examination of the upper extremities revealed nothing of note except a peculiar flattening of the left hand, associated with trophic changes in the fingers, due apparently, to the patient's habit of interposing this member between her body and the bed surface, in avoidance of the irritation arising from such contact.

The patellar reflexes were absent bilaterally. The Achilles reflex could not be obtained on the left and was much diminished on the right. There was no determinable disturbance in the tendon reflexes of the arms. The abdominal reflexes could not be elicited, and the plantar response was of the extension type bilaterally but was more marked on the left. There was apparently no ankle clonus, and no disturbance was observed in general sensibility. The reflexes of automatism were much exaggerated bilaterally, and partial incontinence was reported. The pupils were unequal, and reaction to light and accommodation was apparently much impaired, although a definite statement could not be made owing to cooperative difficulty. Mentally, there was marked enfeeblement in all fields. The occurrence of spasmodic laughter was noted and speech, although definitely explosive and monosyllabic in type, showed no frank evidence of dysarthria.

RAPHAEL, Kalamazoo, Mich.

TUBERCULOUS MENINGITIS. GEORGE FRANKLIN LIBBY, J. A. M. A. **75**:1691 (Dec.) 1920.

Early recognition of tuberculous meningitis necessitates a clear conception of the constitutional, as well as of the ocular, symptoms of the disease. A definite description is difficult to obtain from the literature extant. The theories of the mode of tuberculous infection of the meninges as given by various

writers are reviewed, as well as a number of typical cases in which there were the usual symptoms and course. The author reports four cases of tuberculous meningitis in adults, of the type that is usually first recognized by the ophthalmologist because of the ocular symptoms. The onset of the disease is characterized by severe and constant headache, dimness or loss of vision, paralysis of one or more ocular muscles and, in many patients, ophthalmoscopic changes and hyperemia of the retina and papilla. Miosis, mydriasis and optic neuritis are often present.

In the author's experience tuberculous meningitis preponderated in adults and was more common in men than in women. Early in the disease the symptoms were similar to those of eye strain; there was constant persistent headache with dimness of vision, followed by ocular palsies. Ophthalmoscopic examination was usually negative at first but later became positive. Choked disks with marked retinitis and occasional retinal hemorrhages were rarely observed. Tubercles of the choroid were never seen, but a history of pulmonary tuberculosis was present in all cases observed by the author.

A headache of sudden onset and persistent in character in an adult patient with a history of pulmonary tuberculosis either active or quiescent, should awaken suspicion of tuberculous meningitis, especially if associated with ocular palsy or impairment of vision.

OTT, Rochester, Minn.

LES ALTERNATIVES D'EXCITATION ET DE DEPRESSION, DYSTHYMIE CONSTITUTIONELLE ET PSYCHOSE PERIODIQUE (ALTERNATIVE EXCITATION AND DEPRESSION, CONSTITUTIONAL DYSTHYMIA AND PERIODIC PSYCHOSIS). R. BENSON, *Rev. Neurol.* **27**:30 (Jan.) 1920.

Benon concludes from his study that the characterization "alternative excitation and depression" is loose and clinically inexact. Thus, one must, he points out, in the consideration of "excitation" states, differentiate between five primary types: true manic excitement or "hypersthenia," excitation dependent on "joy" or elation, "anxiety" excitement, "choleric" or rage excitement, and confusional excitement. In contrast to the manic or hypersthenic condition, the depressed state is to be regarded as essentially asthenic in basis, although it may occasionally be found to be dependent on hypothyria or apathy. Benon finds this psycho-physiologic point of view essential to proper comprehension of the depression concept, and in that sense, superior to the older affective or mood conception by which it was made synonymous with "melancholia" in its Kraepelinian significance.

Alternative excitement and depression, while determinable in almost all psychopathologic states, is primarily characteristic of constitutional dysthymia and periodic dysthenia (periodic psychosis of Ballet; manic-depressive insanity of Kraepelin), becoming manifest in the former as a function of the operation of extrinsic stimuli, and in the latter, apparently without objective cause, as a result of intrinsic psychic constitution or make-up.

The author feels the application of the term cyclothymia as synonymous with periodic dysthenia, a misuse, indicating that it should be employed solely to designate the "periodic dysthymias" (periodic anxiety, periodic rage, etc.).

RAPHAEL, Kalamazoo, Mich.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

*Meeting at the Peter Bent Brigham Hospital, Jan. 20, 1921**

GEORGE A. WATERMAN, M.D., *President*

TWO CASES OF TUMOR OF LEFT SUPRAMARGINAL GYRUS WITH APRAXIA. DR. FRITZ BREMER.

CASE 1.—Reported in full in this issue, p. 663.

CASE 2.—The second patient was admitted only a week ago with complete global aphasia, right hemiplegia, apraxia, and mental symptoms, in addition to marked general pressure syndrome of tumor. He was operated on three days ago, at which time an enucleable tumor, as in the former case, was removed from the left supramarginal gyrus. It proved to be a glioma about the size of a tennis ball. There was very rapid postoperative recovery from practically all the preexisting symptoms. The day after operation the patient's mentality was normal, his paralysis had largely disappeared, speech was almost completely regained, and there was but a slight trace of apraxia.

Photographs, diagrams and roentgenograms of the cases were shown.

EXPERIMENTAL DIABETES INSIPIDUS. Presented by DR. PERCIVAL BAILEY and DR. F. BREMER.

This preliminary report concerned the experimental production of prolonged states of high-grade polyuria by minute lesions of the hypothalamus (post-infundibular region). These lesions were made not by the cruder methods heretofore employed, but by bringing the region directly under observation by means of lateral craniotomy. The observations were made on dogs.

DISCUSSION

The discussion was devoted largely to the differentiation between polyurias supposedly of pituitary origin and those due to a lesion of the third ventricle. It was conceded that Dr. Bailey's experiments for the first time conclusively showed that in the production of diabetes insipidus a center at the floor of the third ventricle rather than a disturbance of the hypophysis was at fault. Dr. Bailey was unable to say whether this form of experimental diabetes insipidus could be controlled by injection of pituitary extract.

A CHOLESTEATOMA OF UNUSUAL SIZE REMOVED AT OPERATION. Presented by DR. CUSHING.

The patient was an army officer, who had served in France and who was supposed to have a psychoneurosis. He had not even been a tumor suspect.

* Held on the invitation of Dr. Cushing.

He was an alert and capable officer, the only complaint of his superiors being that he was inclined to assume responsibilities to which he was not entitled. Consequently he had been brought before army examining boards. Various diagnoses had been made; e. g., dementia praecox; general paresis.

A roentgenogram of the skull showed a sharply delimited area of bone absorption in the parietal region, measuring 9 by 8 cm. In one or two places the bone was completely absorbed, and a needle introduced through one of these points of absorption revealed no fluid, though the roentgen-ray appearances suggested a large cyst.

At operation an enormous extradural cholesteatoma, the size of a fist, weighing 175 gm. was removed. The astonishing features of the case were the presence of such a huge tumor, probably of many years' duration, deforming the left hemisphere to an unusual degree without producing any notable symptoms.

Stereoscopic roentgenograms, the tumor, photographs and diagrams were shown. (The case is to be reported in full.)

RESORPTION OF THE CEREBROSPINAL FLUID BY THE CHOROID PLEXUSES UNDER THE INFLUENCE OF INTRAVENOUS INJECTION OF HYPERTONIC SALT SOLUTIONS. Presented by DR. FREDERICK FOLEY (by invitation).

Dr. Foley reported the results of experimental and clinical studies of the effect of hypertonic solutions on cerebrospinal fluid pressure, absorption of the fluid and on brain volume. By animal experiments (carried out in collaboration with Dr. Tracy Putnam, a report of which was published in the *American Journal of Physiology* **53**:464-476 [Oct.] 1920) it was found that gastro-intestinal doses of hypertonic solutions were effective in reducing the pressure of the cerebrospinal fluid and diminishing the volume of the brain. The solutions were as effective by this route as when given intravenously. The medullary depressant effects of intravenous doses were avoided by this route.

In an extension of this work other experiments, in collaboration with Dr. Bailey, were undertaken in an attempt to analyze the details of the mechanism by which the changes were produced. By occipito-atlantoid puncture the sub-arachnoid space was connected to a manometer containing a solution of iron, ammonium citrate and potassium ferrocyanid. Following doses of hypertonic salt solutions the manometer showed the usual fall of cerebrospinal fluid pressure. Coincidentally the ferrocyanid solution was displaced from the manometer into the subarachnoid spaces. The later precipitation of Prussian blue from the ferrocyanid solution showed the solution not only to have penetrated the usual channels of absorption—the arachnoid villi and sheaths of the nerves—but it was also found in the perivascular spaces of the brain and within the ventricular system. The deposit of Prussian blue indicated that the solution had passed through the ependymal epithelium of the choroid into the vessels of the plexus.

In order to exclude mere aspiration of fluid due to enlargement of the ventricles, the manometer was connected to a catheter introduced along the aqueduct, thus separating the ventricular absorption from that in the subarachnoid spaces. The volume of fluid absorbed under these circumstances was greatly in excess of any possible volume change in the ventricular system. The conclusion drawn was that the hypertonic solution by increasing the osmotic value of the blood induced absorption of the fluid from the perivascular spaces of the brain. This withdrawal of fluid from the perivascular spaces causes a

retrograde passage of cerebrospinal fluid from the subarachnoid spaces into the perivascular spaces and finally into the capillaries of the brain substance. Also, there is a retrograde passage of fluid from the subarachnoid space to the ventricular system where reabsorption through the choroid plexus occurs.

The same changes were shown to occur in the human subject; a fall of fluid pressure following hypertonic solution was found by measuring the pressure at lumbar puncture; diminution of brain volume was observed in a number of patients who had developed cerebral protrusion following decompression operations. The volume change of the cranial contents, together with the coincident pressure changes, were measured by an apparatus consisting of a water manometer connected to a pressure bag lying under a plaster cast of this region of the head. The protrusions due to lesions obstructing the ventricular system and accompanied by great dilatation of the ventricle showed such extensive decreases in volume that they could only be explained by retrograde absorption

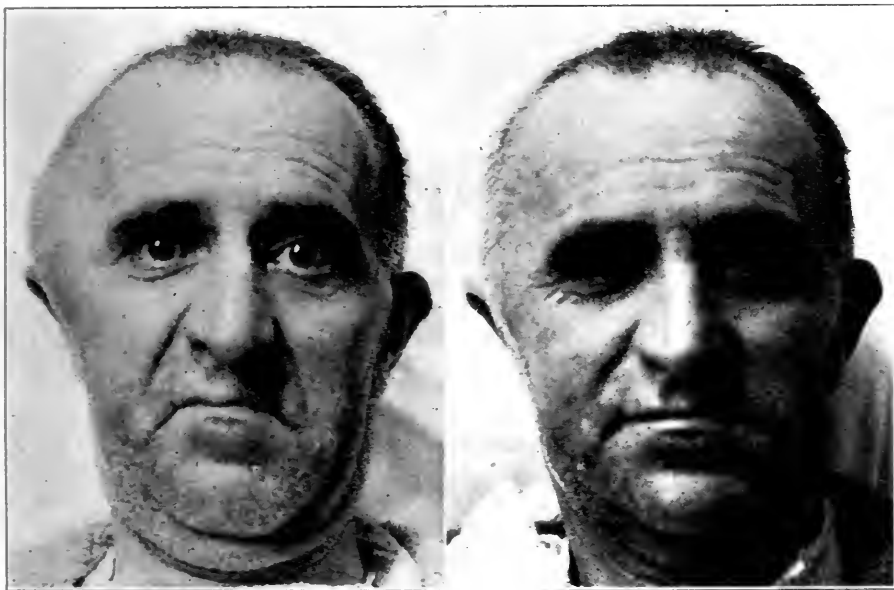


Fig. 1.—Protrusion just before the injection of the hypertonic solution compared with the condition one hour later.

of fluid through the choroid plexuses from within the ventricles. Protrusions unaccompanied by ventricular dilatation showed less extensive volume changes though the pressure decrease was well marked.

The procedure has been employed clinically for the reduction of increased intracranial tension. Photographs illustrative of the results were shown.

DISCUSSION

DR. CUSHING proposed trying out Dr. Foley's conclusions before the meeting by a test case. A patient was shown who exhibited a protrusion through a subtemporal decompression, and in whom an internal hydrocephalus was known to exist. Following the intravenous injection of 15 per cent. sodium chlorid solution, the protrusion completely collapsed (Fig. 1).

A CASE OF MOTOR APHASIA IN A LEFT-HANDED INDIVIDUAL.

Presented by DR. CUSHING.

This patient (Surgical No. 13667), a brain tumor suspect, was shown in view of the question of localization, and to consider whether in left-handed persons the speech center is in the right or left hemisphere. The patient first entered the hospital a month ago with practically no positive neurologic findings except high-grade choked disk and headaches which had been present for a few months. He was accompanied by his brother who observed while here, for the first time, that the patient acted a little peculiarly. He had sent a few checks home for small amounts, but he had made them out on banks in which he had no deposit; an irregularity which in the patient's case was quite abnormal.

As there were no definitely localizing symptoms and as the patient was left-handed, though he had learned to write with his right hand, a left subtemporal decompression was performed, Dec. 10, 1920. He did very well following this operation. The headaches disappeared and there was a rapid subsidence of the choked disk. He left the hospital about three weeks later.

He reentered the hospital a few days ago, owing to aphasia which had come on rapidly in the course of a few days. The area of subtemporal decompression in the interval had gradually become tense.

The patient was presented walking into the room, looking like a normal person, quick and alert in his actions, but could say only, "I don't know," and in his efforts to find words would impatiently snap his fingers and show his concern. He was unable to name the simplest objects, but was able to write with his right hand, though somewhat illegibly, although he wrote his own name freely and well.

Dr. Walton asked how he would sharpen a pencil, and he was given a pencil and a penknife. He took the knife in his left hand to sharpen the pencil—striking evidence of his left-handedness.

Dr. McDonald thought that left-handed people were right-brained only when left-handedness was familial, and not when it was sporadic, as in the case of the patient.

Dr. Bremer said that as a result of his studies of war wounds Marie had come to believe that even in left-handed people the speech centers were in the left hemisphere.

In the patient's case the lesion must be left, for it was apparent to all that there was slight weakness of the right side of the face on emotional movements, and the patient with some difficulty made it clear by pantomime that he felt there was something wrong with his right hand, and held up five fingers to indicate that this had been so for the past five days. The conclusion was that in this left-handed individual, at least, the speech center was in the left hemisphere and the aphasia was of purely motor type.¹

1. The patient was operated on Jan. 22, 1921, an osteoplastic exposure of the left hemisphere being made. The dura was under great tension. Intravenous salt solution was given before it was opened. A tense hemisphere was exposed, with marked convolutional flattening. Although palpation revealed no lesion, a needle was inserted on the chance of finding a cyst. At the base of the second left frontal convolution and at a depth of 2 cm. a gliomatous cyst containing 70 c.c. of straw-colored fluid was struck. The fluid was evacuated, completely relieving the tension. Air was reinserted in the cyst cavity and an immediate roentgenogram taken to show the exact situation of the lesion.

A DOUBLE LESION ACCOUNTING FOR AN UNUSUAL NEUROLOGIC SYNDROME. POLYNEURITIS ASSOCIATED WITH A GLIOMATOUS CYST OF THE CEREBRUM. ROENTGEN-RAY LOCALIZATION OF THE CYST BY INJECTION OF AIR. Presented by DR. GILBERT HORRAX.

History.—E. A. W. A. (Surgical No. 13694), a white girl, aged 9 years, entered the Peter Bent Brigham Hospital, Dec. 17, 1920, complaining of headaches, vomiting and tremor of left hand. The family and personal histories were negative as regards the present illness.

In March, 1920, she became dull in her school work, was irritable, and did not use her left hand so well. One month later she contracted mumps, and after an illness of six weeks the tremor of her left hand became more marked, and she had twitching of the right eye and the right corner of the mouth. She was put to bed, and gradually the left foot began to invert and both feet became weak with the toes in extension. She was taken to a neighboring hospital (Children's) in August, 1920. At that time both legs were almost totally paralyzed, with feet in extension. The deep reflexes were lost at the ankles and knees. There was pain over the nerve trunks in both legs, especially marked in the calves. The right hand was used normally; the left hand was weak and showed marked tremor, with extensive oscillations in attempting to perform any function. The hand had also a static tremor, so that the child had to use the right hand to hold the left one quiet. Impaired sensation to all forms of stimulation was present over both legs, and in addition there was painful hypesthesia over the left hand and arm. Fundi showed at first blurring of the nasal margins of both disks, and later slight choking of the disks. There was left homonymous hemianopsia. The left pupil was larger than the right, and there was marked lateral nystagmus to the right and to the left.

The spinal fluid showed on one occasion 11 cells per c.mm., and on another 13 cells. The blood and spinal fluid Wassermann reactions were negative. Von Pirquet's reaction was negative for human and bovine forms.

The condition was regarded as a polyneuritis. The patient was given braces for her legs, and as the paralysis began to clear up she was gradually able to get on her feet again. She was finally discharged, and told to report at intervals for observation of her eyegrounds.

She reported to the Peter Bent Brigham Hospital in November, 1920, and again in December. At the later date it was learned that the patient had begun to have headaches and vomiting, and had had a seizure which may have been a mild convulsion. Her fundi showed increase in elevation of the optic disks, so she was advised to enter the hospital.

Physical Examination.—This revealed: paresis of the left arm, face and leg, associated with loss of deep reflexes in both legs and the increase of the deep reflexes of the left arm. Both legs were much improved over their condition of four months previously, so far as strength was concerned, as the child could now stand and take a few steps. Tremor of the left hand and arm persisted as marked as before, and similar tremor was present in the left leg.

There were no sensory changes. Nystagmus persisted to the right and left, as did the left homonymous hemianopsia. There was choking of both disks with elevation of 2 to 3 diopters on either side. Pain over the nerve trunks of both lower extremities persisted. The roentgenogram of the skull showed marked separation of the sutures and thinning of the inner table.

The picture presented, therefore, was that of a peripheral lesion of the nervous system, which was clearing up, associated with a central lesion which was getting worse. In view of the advancing choked disks and the left hemianopsia, which could not be explained on the basis of a peripheral disturbance, an exploratory operation of the right hemisphere was undertaken.

This operation disclosed a gliomatous cyst of the right postcentral region. The cyst was largely evacuated, and then filled with air, in order that its extent might be made out by the roentgen ray. Plates taken after the com-



Fig. 2.—Postoperative roentgenogram showing the outline of the bone flap, the anterior leg of which was made in the coronal suture which had been greatly distended. The subtemporal defect is shown by the light area, two silver clips being present in the field. The larger area of the air-containing cyst, measuring on the plate 9 cm. by 5.5 cm., is shown.

pletion of the operation showed a large cyst, running downward and probably inward, about the size of a duck's egg (Fig. 2).

Four days later a second-stage operation was undertaken. At this session the air was evacuated from the cyst, and a good transcortical exposure of the

cyst secured. Its walls were retracted, and at its inferior angle a nubbin of solid glioma was seen, about the size of a hickory nut. This nubbin was removed as completely as possible, and closure made without drainage.

The patient made an uncomplicated recovery, and there was immediate amelioration of all her symptoms. Most noticeable was the improvement in the tremor; the choked disks also subsided. Two weeks after the second operation she was discharged, and was walking about with slight assistance.

DISCUSSION

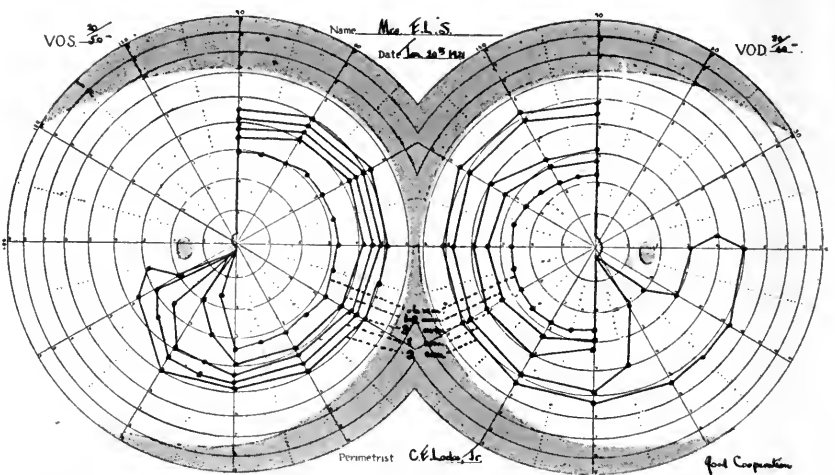
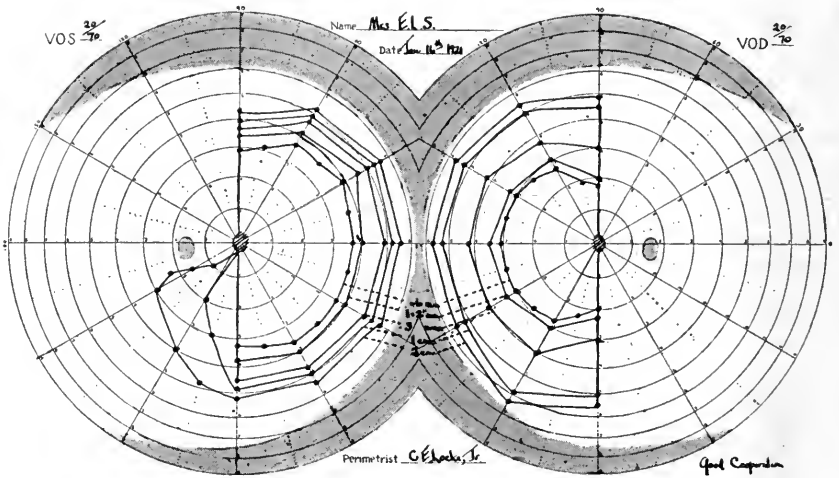
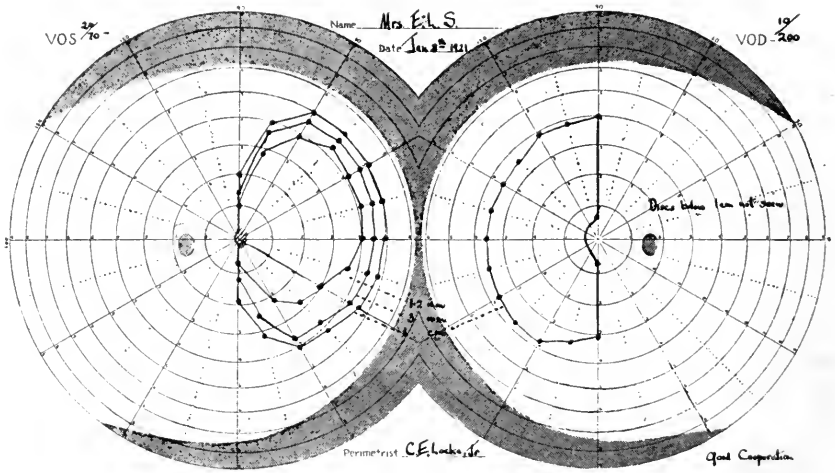
DR. CUSHING stated that for some years attempts had been made to determine the position, size and configuration of gliomatous cysts by the introduction into them of various substances which would cast shadows by the roentgen ray. Since Dr. Dandy's interesting observations at the Johns Hopkins Hospital on ventriculography in cases of hydrocephalus and tumor, air had come to be utilized in the Brigham clinic for this same purpose in connection with cysts. Though Dr. Horrax got into the cyst at the first exploration it was at a depth of 4 cm., and it was highly desirable that the exact situation of the cyst, which was evidently a large one, should be determined, particularly if it was to be opened and its walls treated by a fixative in the fashion commonly employed here. In short, it was highly important to determine where the cyst was most accessible to a transcortical incision. It is hoped that some day these cystograms may be taken with sufficient clearness to enable one to determine the situation of the gliomatous nodule on the wall of the cyst.

THE TRANSSPHEOIDAL VERSUS THE OSTEOPLASTIC CRANIAL APPROACH FOR PITUITARY ADENOMAS. Presented by DR. C. E. LOCKE, JR. (by invitation).

Two patients were shown seven days after transsphenoidal operation. Neurosurgeons do not yet agree as to the comparative value of the two procedures.



Fig. 3.—Sella of patient in Case 1.



Figs. 4, 5 and 6.—Perimetric charts of the fields of vision of Mrs. E. L. S. before and after operation.

The idea seems to be prevalent that a transsphenoidal operation is difficult; that is, dangerous from the possibility of meningitis; and that if successful it may be months before there is any definite improvement in vision. It is the experience of this clinic, the only place apparently in which transsphenoidal operations are done in any considerable number, that the reverse is distinctly the case.

It must be clearly understood, in the first place, that a transsphenoidal operation is only suitable when there is a distended sella easily approachable from below. This is a matter determinable by the roentgen ray. When the sella is small and the neighborhood symptoms are obviously due to a suprasellar tumor, an operation from above is undoubtedly necessary.

During the year 1920 there have been twenty-seven patients with varying degrees of dyspituitarism of one sort or another associated with tumor affecting the chiasm. Of these, fifteen were operated on through the nose and twelve

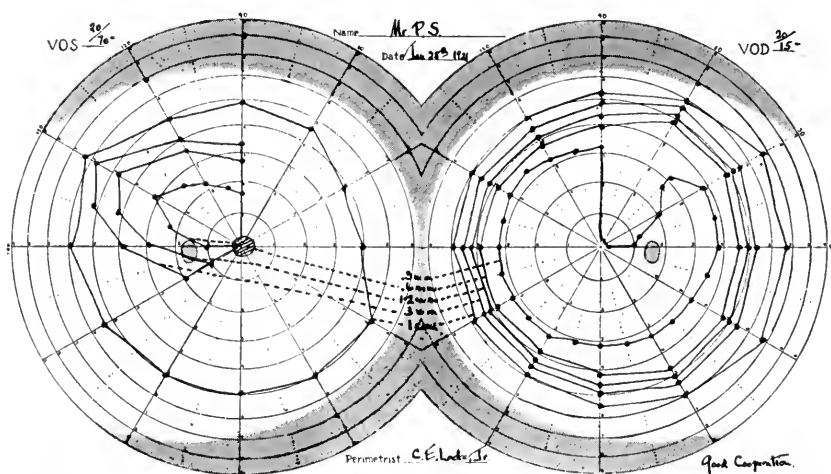
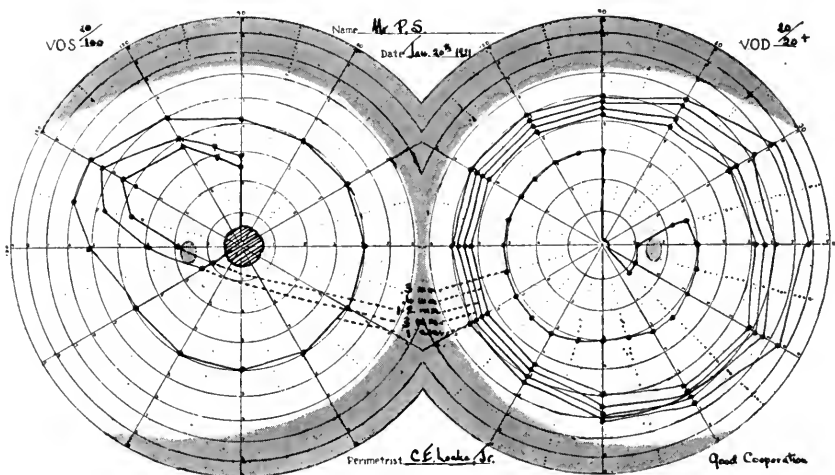
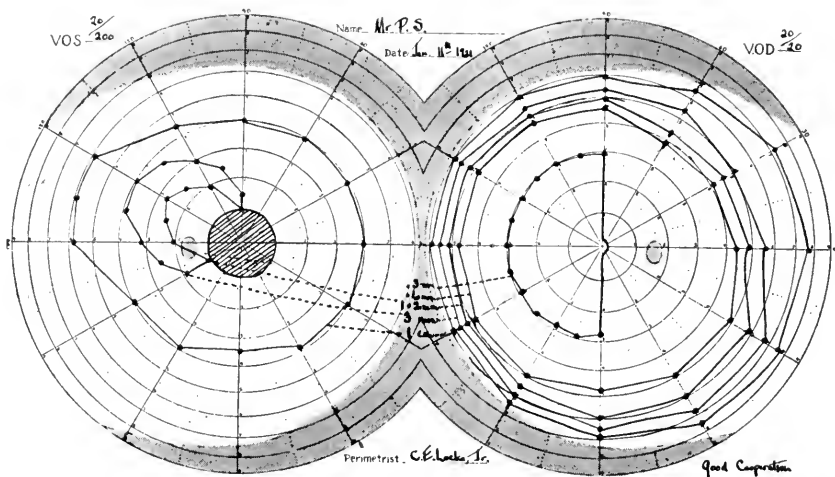


Fig. 7.—Sella of patient in Case 2.

by the transfrontal route. There were no fatalities in either series, but the important fact is that there has been marked and rapid improvement in vision with but one or two exceptions in the group of transsphenoidal cases.

In severity and magnitude the transsphenoidal operation is not to be compared with the osteoplastic procedure, whether from the front or side, and the latter even in the best of hands is admitted to have a high mortality. There is no reason to believe, moreover, that granting a successful attack on the intrasellar tumor, the restoration of vision is not so likely to be just as enduring in one case as in the other. Certainly the risks are much less from below. In the last fifty transsphenoidal cases there has been but a single death.

A criticism of the transsphenoidal procedure has been made, and perhaps justly, that it will not reveal a possible intracranial extrusion of the tumor through its dural capsule, a condition which could not possibly be dealt with from below. Leaving aside the possibility and probability that neither can such



Figs. 8, 9 and 10.—Perimetric charts of fields of vision of Mr. P. S., immediately before and after operation.

an intracranial extension of the lesion be thoroughly eradicated by an operation from above, the real answer to this criticism lies in the fact that an extrusion of the tumor through the sellar base into the sphenoidal cells is inaccessible from above. It is a condition shown by the first of these patients.

CASE 1.—Mrs. S. (Surgical No. 13793), aged 60, referred by Dr. B. A. Cohoe of Pittsburgh, entered the hospital January 6, complaining of loss of vision. From the appearance of her skin a pituitary disorder might be surmised, but the degree of dyspituitarism was not great. Her basal metabolism on entrance was -6 . The chief signs of trouble related to her sella and vision. The sella was of the greatly distended type (Fig. 3), and stereoptically it was evident that it protruded far into the sphenoidal region. Quantitative perimetry, as introduced into this clinic by Dr. Walker, showed a bitemporal defect. With the right eye disks smaller than 1 cm. could not be seen, and there was a central scotoma, vision being reduced to shadows 10/200. With the left eye the 1.2 mm. disk was the smallest the patient could see, vision being 20/75.

At operation a soft adenoma was found completely filling the sphenoidal cells, having burst through the sellar base and dural capsule of the enlarged gland. The cells were completely cleaned of the adenomatous masses (shown to be a chromophobe adenoma by Dr. Bailey). The remains of the distended sellar floor were removed and a large portion, possibly one half, of the intrasellar gland removed. She made a perfect recovery. There is now no nasal discharge whatsoever and from an intranasal examination one would not know that any operation had been performed, the septal mucous membrane being, of course, intact.

Fields of vision were taken on the third day and again this morning (Figs. 4-6). They are widening rapidly, but the chief improvement is in the visual acuity which has increased in the right eye from 10/200 to 20/40. Mrs. S. is anxious to return home. However, before doing so she should have a course of deep radiation with the roentgen ray or a local treatment or two with radium emanations.

CASE 2.—Mr. S. (Surgical No. 13802), aged 52, was referred by Dr. H. L. Sloan of Charlotte, N. C., the chief complaints being headache and loss of vision. The evidences of dyspituitarism were more marked in this case, and his basal metabolism on entrance was -21 with subnormal temperature and pulse.

The neighborhood signs were not unlike those of the first case. The sella (Fig. 7) was greatly widened, with absent posterior clinoids. There was primary optic atrophy. The quantitative fields showed a tendency to homonymous defect (Fig. 8). To the 1 cm.-disk, usually employed alone, the hemianopsia on the right, where vision was unaffected, would not have been apparent, for this was only brought out by the smallest visible disk -0.3 mm. On the left there was a large central scotoma, and disks below 1 cm. were not seen in the nasal fields. The vision in the left eye was 20/200.

The operation, performed seven days ago, was conducted without difficulty. The thin bulging floor of the sella was easily brought into view on removing the anterior wall of the sphenoidal cells. The thin rubbery sellar base was removed, the dura incised, a generous intrasellar removal of possibly the lower half of the adenoma was made (chromophobe struma: Dr. Bailey).

This patient did even better than the former patient. There was immediate subjective improvement in vision in the left eye, and the perimetric charts taken on the third day and again this morning show considerable widening of the fields, which will undoubtedly soon be quite normal on the right, though

on the left less may be expected as the nerve has been far more seriously damaged. Vision, however, has already improved from 20/200 to 20/70—.

Mr. S. is able to go home, but will remain another week for transsphenoidal radiation of the glandular region.

A CASE OF PULSATING EXOPHTHALMOS. Presented by DR. YOAKAM (by invitation).

Though this condition is easily recognizable, it presents an extraordinary picture which makes the unfortunate victims objects of interest and sympathy.

History.—Robert T. (Surgical No. 13799), a schoolboy, entered the hospital, referred by Dr. J. R. Eastman of Indianapolis. On May 8, 1920, when 13 years of age, he was shot by a 22-caliber rifle, the point of entrance being the right mastoid process. He was unconscious for only a few minutes and managed to walk home but was drowsy for twelve hours. On the following day he had palsy of the right side of the face, which cleared in a few days. For three weeks he had severe headaches, after which a subjective bruit developed. This



Fig. 11.—Pulsating exophthalmos from gunshot wound.

was followed by marked exophthalmos, abducens palsy, and great dilatation of the vessels about the right orbit. Thus it took three weeks from the time of the injury for the anastomosis to form (Fig. 11).

Examination revealed the phenomena attendant on an arteriovenous aneurysm, but the communication must be extracranial judging from the position of the bullet just below the base of the skull.

PRESENTATION OF CASES.

The following series of cases from the hospital wards were briefly presented for diagnosis and discussion. Most of them were cases sent in for study as tumor suspects. The method of classifying tumor cases which has been adopted on the surgical service was described: namely, into (1) *tumors verified* by histologic examination of tissue; (2) *tumors not verified*, though undoubted and perhaps even exposed at operation; (3) *tumor suspects* where there may be considerable doubt though the case had been regarded as one of presumptive tumor by the recommending physician.

CASE 1 (Surgical No. 13870).—A patient with undoubted multiple sclerosis.

It is hardly conceivable that a case of this kind, particularly in the absence of choked disk or other pressure symptoms, should even for a moment be seriously regarded as a possible cerebellar tumor. Yet the patient was referred with this diagnosis. The patient showed marked nystagmus and extraordinarily marked incoordination of the extremities, of such degree that gait and station could not be tested. Hypotonia of the extremities was extreme. There was also considerable dysarthria.

CASE 2 (Surgical No. 13851).—*A patient with focal epilepsy, brain tumor suspect: presumably endothelioma.*

The patient, a man aged 41, gave a history of generalized severe headache for the past twenty years. Two months before admission he awoke with a feeling of weakness and awkwardness in his left hand. A few days later he had a typical jacksonian attack beginning in the left hand. There had since been four similar seizures but never with loss of consciousness. For the past ten days there had been frequent minor attacks of numbness limited to the hand. The reflexes on the left were slightly exaggerated. There was no choked disk. The roentgen rays showed no change in the skull.

Even in the absence of objective evidences of pressure this was unquestionably a tumor, and if we are to regard the headaches as occasioned thereby it was probably a benign lesion and possibly an endothelioma. However, were this so one would expect some evidences in the skull, either a point of thickening (endostosis) or dilatation of the diploetic vessels.

[This patient was subsequently operated on and a postcentral glioma disclosed. The lesion was enucleated. A defect exactly corresponding to its position was then made in the bone flap before its replacement. Deep radiations (roentgen) were subsequently given in the direction of the lesion, through this defect.]

CASE 3 (Surgical No. 13833).—*A cerebellar tumor suspect: illustrating the effect of pregnancy in accelerating tumor growth.*

The observations on mouse tumors have shown that at the termination of pregnancy there may occur a marked increase in the growth curve of these lesions. This observation may perhaps explain why it is that the onset of so many cases of brain tumor in women appears to coincide with pregnancy.

This young woman entered the hospital on January 13. Last July when eight months along in her first pregnancy, she noticed dizziness on turning suddenly. She had been vomiting throughout the entire period though she had been without headache. Later in July progressive loss of hearing on the right was observed. Soon after parturition, which was normal, she noticed numbness of the right side of the face, and occasional numbness of both arms; in September, unsteadiness with swaying to the right. Early in December, stiffness of the neck and severe headaches came on and choked disk was observed.

Examination.—The positive findings were: Choked disk of 5 diopters with typical chrysanthemum nerve head; nystagmus more marked to the right; static instability with deviation to the right; incoordination chiefly in right leg and arm; lowered reflexes with hypotonia on the right; hypesthesia of the right trigeminal skin field; suboccipital tenderness; lowering of auditory acuity on the right.

Though she did not have a typical history, the patient was naturally an acoustic tumor suspect.

[She was operated on January 24. The usual bilateral suboccipital exploration was made. The ventricle was punctured, revealing hydrocephalus. No tumor was disclosed though the search in each recess was carried to the region of the pons. There was a large posterior cistern with a thickened grayish wall, and the case may be one of chronic arachnoiditis. The choked disk subsided rapidly and she was discharged much improved. The diagnosis remained "Cerebellar tumor: not verified."]

CASE 4 (Surgical No. 13783).—*A brain tumor suspect: presumptive diagnosis of cerebral arteriosclerosis and thrombosis.*

It was stated in regard to this patient that one cannot be certain of the diagnosis of tumor or nontumor without operation or necropsy. A working-man, aged 49, with a presumptive history of syphilis and with advanced chorioretinitis in one eye was admitted to the surgical wards on January 4. He gave a history of having been well, except for loss of vision in his left eye, until three weeks before entrance. He awakened one morning with severe left-sided headache. In the course of forty-eight hours there had come on a spreading paralysis of his right side, beginning in the arm and becoming complete. At the same time there was marked aphasia. His condition was such that the sole source of the clinical history was a relative. He had obvious arteriosclerosis, and there were no evidences of his having any special grade of intracranial pressure and no history of headaches. His eyegrounds were negative except for some possible haziness of the nasal margin of the left nerve head, a condition considerably obscured by the extreme degree of patchy pigmentation throughout the retina. His blood was negative, but the Wassermann reaction of the spinal fluid was positive in 1 c.c.

In commenting on this case, attention was drawn to the tendency on the part of the hospital physicians to make, when possible, a presumptive diagnosis other than tumor, whereas the reverse was the natural tendency on the part of the surgical staff.

A patient with a closely similar history had, shortly before, been on the medical service with the diagnosis of cerebral thrombosis in the absence of choked disk. The patient died and came to necropsy. The clinical history was used in Dr. Richard Cabot's third-year exercise, and both he and the class arrived together at the diagnosis which had been made in the wards during life. The necropsy, however, disclosed a large glioma.

This experience shows that it may sometimes be difficult to distinguish tumor and thrombosis, but nevertheless, the latter is the more preferable diagnosis in this case, and treatment for syphilis will be instituted.

[The patient shown at the meeting as a presumptive vascular case, some days later became deeply stuporous and a reexamination of the fundus showed that there was definite swelling of the left nerve head of about 2 diopters. Under local anesthesia, on January 17, a left subtemporal decompression was performed by Dr. Locke, revealing great tension. He made a good recovery and regained consciousness. On February 5, Dr. Horrax made an osteoplastic exploration, revealing a large apparently enucleable tumor of the postcentral area. Five days later this tumor, which proved to be a neuroblastoma, was enucleated. This led to prompt improvement in the paralysis and the faculty of speech was entirely regained.

The experience well illustrates the reason why we have come to keep a separate list of brain tumor "suspects."]

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Feb. 17, 1921

JOSEPH W. COURTNEY, M.D., *in the Chair*

ANATOMIC FINDINGS OF GENERAL PARESIS AND MULTIPLE SCLEROSIS IN THE SAME CASE. DR. SOLOMON C. FULLER.

Dr. Fuller discussed an association of typical cerebral lesions of paresis with multiple sclerosis lesions, the question being whether there was an actual combination of paresis and multiple sclerosis or whether the multiple sclerotic areas were manifestations of a disseminated nonparetic syphilitic process.

A woman, aged 47, presented definite indications of paresis both mentally and physically and also certain signs strongly suggestive of multiple sclerosis. For a greater part of the time toward the end of her illness she was apparently obliged to use crutches; on occasions of excitement and euphoria she was able to discard them and even to run. This had led to the supposition, on the part of her friends, that her disability was more imaginary than real. Histologic examinations of the cortex showed alterations typically parietic. Preparations of the chiasm, oblongata and cord showed disseminated sclerotic areas indistinguishable from the typical lesions of multiple sclerosis. A careful search failed to reveal spirochetes.

DISCUSSION

DR. H. I. GOSLINE asked whether Dr. Fuller had considered the possibility that these changes in the spinal cord which show no sign of inflammatory process may be later stages in the natural history of the syphilitic process.

DR. S. C. FULLER replied that it might be possible. The late stages of a multiple sclerosis are indistinguishable from any chronic encephalitis with fibrous replacement. However, no fresh multiple sclerotic lesions were encountered in this case.

A CASE OF HENSCHEN'S NODES. DR. JOSEPH W. COURTNEY.

In 1881, Henschen wrote a paper on migraine in which he stated that 106 out of 140 patients showed nodes or nodules scattered about either in or under the skin of the arms, neck and scalp. Dr. Courtney said that for a long time he had been looking for such nodules and had found them in only one patient, whom he exhibited. She had had classic migraine for many years. A year or more ago she noticed nodules under the skin, in the arms and sternal region but none in the scalp or neck. They were more sensitive at some times than at others.

DISCUSSION

DR. E. W. TAYLOR thought this interesting in connection with Auerbach's observation of what he called "nodular headaches." In many cases of headache, he had observed nodules in the muscles about the neck and shoulders, which were rather easily removable by massage. Such headaches he sharply distinguished from those of the migraine type.

PERIPHERAL NERVE INJURIES TREATED AT THE MASSACHUSETTS GENERAL HOSPITAL. DRs. W. E. PAUL and C. A. PORTER.

Dr. Paul reviewed 188 cases, from 1878 to 1918, involving fourteen nerves injured singly or in combinations and including brachial plexus injuries. Of these, ninety-five had a record of end results. The pioneer operation was

done in 1878 by the late Dr. C. B. Porter—a primary suture of the ulnar and median above the elbow. The operative wound suppurated, as did most of the nerve sutures in the preaseptic days. The injuries showed thirty-four kinds of trauma or conditions and twenty cutting agents were specified, glass predominating. Nerve inclusions occurred in three forms. The nerves most frequently cut were ulnar, 31 cases; median, 18 cases; ulnar and median, 12 cases; musculospiral, 8 cases; external popliteal, 4 cases. In operations various methods were used to approximate nerve ends and favor healing. Excision of portions of long bones was twice practiced. Frequently the sutured nerve was wrapped in Cargile membrane, or fascia, or fat or muscle. A vein was used to guide growth over a gap. Heterotransplants and autotransplants were resorted to. Implantation of a neighboring nerve was done. Gaps were bridged with strands of silk, etc. Several patients were operated on more than once.

Males were found to preponderate in numbers injured. There were ninety-five cases with a record of the condition one year or more after operation. In the sixty-five suture cases, 41 per cent. failed to improve; 26 per cent. showed slight improvement; 30 per cent. showed considerable improvement; one patient (1.5 per cent.) was stated to have completely recovered. In the thirty neurolysis cases, 40 per cent. showed no improvement, 23 per cent. slight improvement, 20 per cent. considerable improvement, 16 per cent. complete recovery. These studies, though giving little help in treatment, demonstrate how much repair is required for complete restoration of function following suture of a divided nerve. Surgical and neurologic opinion seems to be settling on three apparently sound factors essential to repair that shall restore function: (1) accurate apposition by end-to-end suture of the central divided end to its former glial pathway peripherally; (2) a technic to avoid meddlesome handling of the nerve and eliminate postoperative fibrous tissue growth or clot at the nerve junction; (3) methods of suture to minimize operative trauma.

DR. C. A. PORTER stated that up to the time of the war, there had been no one who had had a large experience in such civil surgery as most of the patients came to the hospital with a history of having been previously treated by some emergency surgeon who, not infrequently, sewed up the tendons omitting to suture the nerves, or sutured the nerves with chromic catgut, silk or big needles. In such injuries as badly cut wrists, unless adequate experience and proper surroundings were available, it would be advisable to stop the hemorrhage, render the wound as clean as possible, dress with a sterile dressing and send the patient to the nearest competent surgeon to deal with the nerve injury. This applies particularly to injuries of the ulnar nerve which presides over the fine movements of the hand and fingers. It is doubtful whether a primary suture of the ulnar nerve ever results in perfect motor and sensory return, and it is almost sure that a secondary suture never so results. When there has been a considerable lapse of time between the injury and suture, a varying degree of contraction, deformity and degeneration invariably preclude complete restoration of function. Even when there has been perfect healing after primary suture and the patients have had regularly the kind of after-care which they obtain in the outpatient department, the result, when tested carefully, is never perfect.

The sciatic nerve, on the other hand, presides over rather gross movements and comparatively unimportant sensations, so that when the toe drop is corrected, it is surprising to see how well these patients can walk. When the sciatic nerve or its branches have been divided or a tumor excised, it is often

difficult to bring about end-to-end union without undue tension. Much can be gained, however, by proper, long continued position of the limb. The higher the suture and the larger the defect, the longer should the limb be immobilized.

A severe injury to the brachial plexus, of all peripheral nerve lesions, requires the nicest judgment as to what should be done. In a total of twenty-seven personal operations, only one has given satisfaction, and this was a comparatively poor result although the patient had been treated as a private patient by one of our neurologists for over three years. As a five-year end result, she could flex her arm, wrist, fingers and thumb. The lesion was originally complete, and the operation was a combination resection end-to-end suture and lateral anastomosis. In almost all of the other cases, in spite of endeavors to put sound nerve to sound nerve, the results have been lamentable and have almost always ended with shoulder amputation. In a small number of these cases, on account of persistent pain, posterior nerve-root division has been necessary. Of these patients only one was cured, the failures probably being due to inadequate division. Dr. Porter advised resection of the roots from the third cervical to the first or even second dorsal. If only the roots of the brachial plexus proper are divided, there is invariably a return of pain.

In regard to Volkmann's paralysis, among the patients operated on, two or three clearly showed that in addition to marked atrophy and fibrosis of the muscles, there was a definite lesion of the nerve as well, and in one case, above a point of constriction, a definite neuroma had formed. When the involved muscle has become functionless, no matter whether nerves could be freed by operation from scar tissue or united, the only hope of improvement is by transplanting normally innervated tendons.

In the after-care it is vitally important that the paralyzed muscles be relaxed, as in general one finds that the normal muscles have, sooner or later, brought about contractions most difficult to overcome. Massage and galvanism should be employed to keep up the muscle tone and nutrition, and the finer the function of the nerve, the greater the importance of this after-care.

All are agreed that the musculospiral nerve with comparatively gross function is the one which recovers most quickly and satisfactorily. In many cases neurolysis, after fracture in the middle of the humerus, has proved curative, and, if in doubt, should be tried without resorting to resection. After thorough freeing of the nerve, which should always be commenced from above and below, thus approaching the site of the lesion, the nerve may be stretched, as a nerve with moderate traction will stretch from one-half to one inch, but little or nothing can be gained by pulling the distal end upward. As one's experience increases, however, and the dissection upward is more freely prosecuted, if the adjacent joints are properly relaxed, one can usually obtain an end-to-end suture in the average lesion.

Little advance has been made since 1885 in the actual technic of suture, though probably today the majority of surgeons use absorbable material. Experience has shown the importance of delicate handling of the nerve, of the necessity for absolute hemostasis and the advantage of avoiding rotation in suture in order to maintain the original nerve tract pattern.

Before the nerve is freed, it is flat in the majority of cases rather than round and composed sometimes of many nerve bundles rather than a single cable. Almost without exception there is a good sized bulb on the proximal end, while that on the distal is either much smaller or sometimes absent. Usually a little fibrous cord connects the bulbs. With the wish to remove as little as possible of these bulbs in order to be able to suture end to end,

the human tendency is to inadequate excision of the proximal neuroma. Dr. Porter believes, owing to this error, that many nerve sutures are doomed from the start, and would strongly urge section after section with a fine, very sharp knife until motor nerve cables can be clearly demonstrated. Probably little harm results from placing one catgut stitch through the center of the nerve to approximate the ends grossly, but on this authorities differ. The rest of the suture should be done with very fine chromic catgut or very fine silk, the sutures being placed through the nerve sheath in such a way that the down-growing fibers are directed into the distal end and cannot mushroom out through the suture. This effect, which is not desired, is sometimes produced by drawing the central single stitch too tightly. Dr. Porter believes that the introduction of any foreign body is contrarily indicated. Some physicians have advocated surrounding the suture by fat or fascia lata.

The main question involved is not the technic of the placing of a few sutures to approximate nerve ends, but a question of experience and judgment in selecting the best procedure in any given case. On the other hand, the surgeon must constantly bear in mind that the vital point of perhaps a two-hour tedious operation is the ultimate nerve suture which in itself may not require more than ten minutes.

DISCUSSION

DR. J. B. AYER spoke of a patient whom Dr. Porter operated on about 1911. In this case he had a chance to examine the tissues. The patient had been cut by a piece of glass and a neuroma about the size and shape of a pecan was excised. Histologic examination showed that he did not get above to perfectly normal nerve, as there was a good deal of fibrous tissue at the proximal end. About one-half to one-third of the fibers ran through to the distal end. After resection he could not unite the ends, and there was an inch gap filled in with several silk sutures and surrounded by Cargile membrane. About three years later the patient was able to use all of her ulnar muscles. Sensation had returned to a considerable extent although of a protopathic rather than epicritic type. Dr. Paul would agree that there was not complete regeneration, but it was a very useful hand. This result was obtained under conditions which Dr. Porter has said are the worst: namely, the nerve affected was the ulnar, and the gap bridged was a considerable one.

DR. E. W. TAYLOR remarked that nothing had been said of the method of regeneration of divided peripheral nerves, whether solely from the proximal end or whether in part, at least, from the neurilemma sheaths of the peripheral end. This matter, long in dispute, is of practical importance in connection with the time of suture after the injury.

DR. G. L. WALTON remarked that in view of the "fumbling period," when various devices were tried for regenerating nerves at a distance, it was a great satisfaction to have established the realization that such efforts may be discarded, and that there is little or no result to be expected unless there is absolute apposition of the cut ends.

DR. PERCIVAL BAILEY had recently seen Dr. Dean Lewis's work in Chicago, and felt that it was necessary to have an absolute end-to-end anastomosis without tension if results were to be obtained.

DR. STANLEY COBB spoke of his work under Dr. Frazier in General Hospital No. 11. In six months about 175 patients with peripheral nerve wounds were treated, and the rule was to wait until three months after the last sign of

infection in the wound. That frequently meant waiting until six or nine months after the original injury. On making then an end-to-end anastomosis, it was found that regeneration would at once begin as though the operation had been done immediately after the injury, which is evidence in favor of the theory of central regeneration only.

DR. J. W. COURTNEY called attention to the fact that the electrical excitability of sutured nerves is an absolutely unreliable index of their functionality. He spoke of a young man in whom the left ulnar had been partially severed by a pen-knife. Dr. Porter had done a very careful and successful end-to-end anastomosis. In spite of the slow but steady regeneration of muscles and return of function which followed the nerve anastomosis, neuromuscular electrical excitability remained practically at the zero point for considerably more than a year from the date of operation.

DR. W. E. PAUL, in answer to Dr. Taylor's question, stated that the authorities seemed to be in favor of the central regeneration theory. It is interesting to realize that the degenerative changes in the peripheral segment of a divided nerve include the disappearance of the parenchymatous tissue of the nerve.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 20, 1921

G. B. HASSIN, M.D., *President, Presiding*

ELSCHNIG'S TYPES OF PHYSIOLOGIC DISKS. DR. E. V. L. BROWN.

In the little read (and unfinished) encyclopedia of ophthalmology edited by O. Schwarz, Elschnig of Prague has attempted a classification of optic disk into types based on the amount of connecting and supporting tissue in the nerve trunk. When there is a large amount of this tissue the bundles go straight forward, parallel to a point well anterior to the inner surface of the sclera, then diverge abruptly, almost at right angles and go over into the retina, leaving only a shallow funnel in the center with none of the lamina cribrosa visible at its bottom. The vessels come well forward with the supporting tissue and divide on the surface. This is his Type 1. In Type 2 there is less connective and supporting tissue (pial and glial), and a narrow cylindrical excavation takes the place of the funnel, and the vessels divide as before on the surface. In Type 3 there is marked lack of tissue between the bundles, nothing supports them in front of the lamina cribrosa, they no longer run parallel any distance after they go through the holes in the lamina cribrosa, and they bend away in all directions from the central axis of the nerve trunk and come to lie along the wall near the edge of the scleral canal. This leaves the wide gap or "cup," called a large physiologic cup or excavation. On the temporal side the bundles go only a short way (some 3-4 mm.) to the macula, and these few nerve fibers are spread over five-twelfths of the circumference of the disk, at that, so the amount of tissue between them, too, is small, and the cup comes to be especially large in the temporal part of the disk, on the side of the macula. The artery and the vein both divide on the floor of the

cup. In Type 2 or 1 only the vein divides on the floor. A large amount of lamina cribrosa is exposed to view. This, in the main, explains the plan of the classification.

Any man or any group of men who regularly try to "type" the nerve heads they see will, in Dr. Brown's opinion, find the classification tenable and will find that it adds a great deal of interest to the study of a disk. Its prime value lies in the training it gives. But, if one learns to differentiate between a Type 2 and a Type 3 disk he will never make the error of calling a disk with a large physiologic cup an "atrophic disk." This is quite frequently done by doctors who, Dr. Brown said, he felt had never really seen anything interesting enough about a disk to make them study it intelligently.

DISCUSSION

DR. HUGH T. PATRICK asked whether the different disks and cups made any difference in the appearance of pathologic processes; whether a choked disk would be less obvious in Type 3 and would be later in showing itself. The cup would fill up more slowly, and he wondered whether it would make any difference in the time when an optic neuritis or glaucoma might be recognized.

DR. RALPH C. HAMILL said that in London the neurologists spoke of the swelling of the nasal half of the disk. He asked whether the ophthalmologists consider that there can be a swelling of the nasal half with a fairly good cup.

DR. PETER BASSOE asked whether Dr. Brown could lay down some rule whereby one could distinguish the temporal pallor in multiple sclerosis from various normal types of disk. He recalled many cases in which he thought he saw this pallor, and would have to admit that had he seen a similar disk in a person without any organic disease he would have passed it up as some variation in the disk, which was scientifically dishonest.

DR. BROWN, replying to Dr. Patrick's question as to the incipient stage of the organic atrophy, said he did not think it would make any difference, because the cup was probably less than 0.5 mm. wide. If there was any edema, which was the recent way of looking at all papillitis cases, it would progress across this 0.5 mm. very soon. In the physiologic cup the disk never goes to the very edge. There is always the intervening wall of nerve tissue, even when it is as thin as was shown in the illustration of Type 3. The glaucoma cup always reaches to the edge of the disk at some one point; it may not be for more than one-twelfth of the circumference, but when one is differentiating between the glaucoma cup and the large physiologic cup the course of the vessels settles the question.

Dr. Brown thought the relatively small amount of the nerve tissue that goes to the temporal side in comparison with the amount that goes to the nasal side answered Dr. Hamill's question. Gowers was the first to point out that choked disk and neuritis begin on the nasal side, and that one could have choked disk that involved only that portion in which the tissues were the densest. The surface of the disk and the macula often differ as much as three diopters, 1 mm. Dr. Brown took a section of a disk to Professor Graef, Berlin, and asked him whether it was high enough for a choked disk, but could not get him to say that it really was a normal or a pathologic disk. There can be great variation in the height of the disk. The clouding can usually be seen first on the nasal side.

The most characteristic thing about multiple sclerotic atrophy is that in one eye it may be the superior quadrant and in the other eye another quadrant,

perhaps the inferior quadrant. The atrophy of tabes is regularly more in evidence on the temporal side.

Dr. Brown thought that Dr. Bassoe brought up the point which settles in the main the question as to whether or not the atrophy goes to the edge of the disk. In the patient presented there was a well colored but narrow wall of nerve tissue temporal to the cup. If this patient should develop a temporal pallor, there would be a small amount of tissue involved. There was no end to the amount of dispute regarding the pallor of the disk on the temporal side.

TUMOR OF THE LEFT BASAL GANGLIONS AND CEREBRAL PEDUNCLE. DR. RALPH C. HAMILL.

Mrs. B., 44 years of age, entered Wesley Hospital in June, 1920. Family and previous histories were uneventful. In the summer of 1919 conduct changes were noted. She wanted to take out a great number of insurance policies. Shortly after this she began to have vomiting attacks, with and without headaches. Early in 1920 weakness, gradually developing into paralysis, of the right arm and leg was observed so that by June she was unable to walk. Shortly before entrance she began to have difficulty in finding words and involuntary micturition appeared. At the time of entrance there were slight anemia, normal urine, negative blood and spinal fluid Wassermann reactions. 27 cells.

July 30: Voluntary movements of the upper part of the face were good and equal. The right lower side of the face was paretic, more marked in emotion than volition. The motor fifth was normal, the right corneal reflex diminished. The right eye turned slightly down and in, yet there was no true paralysis. Occasionally there was slight forced movement of the eyes toward the right and possibly paralysis of upward movement of the right eye. There was paresis of the right arm and leg with increased tonus and loss of the sense of passive movement of the right hand. Attention was poor, hence sensory changes were difficult to determine. She seemed to hear better with the left ear. There was marked bilateral choked disk. The patient lay in a semistuporous condition, seldom speaking, and then only in monosyllables or short sentences.

August 4: The patient lay quietly, almost never speaking. She ate when fed, answered simple questions, smiled rather easily, but attention was very poor and she made no attempt to follow any but the simplest instructions. Memory seemed good so far as it could be tested. There were occasional signs of perseveration. The left side of the face tended to smile as soon as she started to speak. It was impossible to make her cry though pin pricking brought tears. She had no anosmia. Marked bilateral optic neuritis, with small hemorrhages, was present. There was no evidence of hemianopia. She tended to keep the left eye closed (apparently to avoid double vision). The right eye turned down and in, though it could be abducted to the outer canthus. There were less upward and downward movements than five days before. Lateral movements were good, though there was slight nystagmus, greater in the left lateral position than in the right. The pupils were irregular and reacted poorly to light. It was impossible to determine reaction in convergence. The motor fifth was normal. The right corneal reflex was decidedly diminished, and slight hypesthesia over the right cheek and forehead was suspected. The right side of the face was paretic, slightly above, more below, more marked

in emotion than volition. She appeared to hear a whispered voice normally. The functions of the ninth, tenth, eleventh and twelfth nerves were normal. The right arm was sharply flexed at the elbow and adducted. The fingers were clenched. She performed all but the finer movements with the fingers and hand, but only with marked effort and very slowly. There was practically no spontaneous movement of the right arm or leg; there was resistance to passive movements of the arm but not of the leg. She seemed to feel pin prick normally. The right plantar reflex was strongly extensor, the left doubtful. There was right ankle and patellar clonus. The right abdominal reflex was absent, the left diminished. Arm reflexes were exaggerated on the right, brisk on the left.

A diagnosis of brain tumor was made, localized in the left cerebrum and causing pressure in the region of the anterior corpora quadrigemina, optic thalamus and internal capsule.

August 15: The patient lay in stupor most of the time, though she could be aroused without difficulty. The muscles of the right hand were atrophic and the thumb tended to take the plane of the palm. Still, coarse movements of the thumb and finger were possible. In the midst of the examination she suddenly looked at the examiner and said quite clearly and slowly, "I don't see why you have to bother me at the last."

September 1 she was found by the nurse in coma, which lasted only a few hours.

October 6, the patient was found lying on her back with the head turned to the right. The right eye was partly open, the left eye closed. The eyes, which were held to the right, were constantly agitated by slow movements toward the midline and quick movements toward the right. Occasionally she turned her chin toward the midposition; then with short jerks synchronous with the quick movement of the eyeballs the chin rotated to the right. The nystagmoid movements of the eyes, more marked in the left one, were about ninety to the minute. There was also a fine oscillating tremor of the left eye. The pupils were equal and reacted to bright light. The corneal reflex was lost on the right, diminished on the left. Paralysis of the right arm and leg was complete. The bilateral Babinski sign was present; right ankle and knee reflexes were absent, the left normal. The right wrist reflex was greatly exaggerated. The right abdominal reflex was absent, the left diminished. She seemed to feel pinching everywhere. There was practically no speech.

This conjugate deviation of the head and eyes lasted only for one day and did not return. The patient gradually became worse, developed *snout-krampf* expression and died, Nov. 28, 1920. Throughout the last three months of life the pulse was always over 100. Respirations were usually from 20 to 24. The temperature was normal until the last few days, when it gradually rose to 105 F., with a terminal pneumonia.

The interesting features of this case are the paralysis of upward movements of the eyes, the motor disturbances of emotional expression and the short period during which there was conjugate deviation of the head and eyes toward the paralyzed side. Landouzy reached the following conclusions concerning conjugate deviation of the head and eyes in apoplexies:

1. If the eyes turn toward the side of the convulsing limbs there is an irritative lesion in the cerebral hemisphere.

2. If the eyes turn away from the paralyzed limbs there is a paralytic lesion in the hemisphere.

3. If the eyes are turned toward the paralyzed limbs there is a paralytic lesion in the pons.

4. If the eyes are turned away from the convulsed limbs there is an irritative lesion in the pons.

Sections of the brain of the patient showed a tumor obliterating the normal markings of the basal ganglions from a point a little anterior to the optic chiasm backward to almost the posterior horn of the lateral ventricle, and extending down into the left peduncle to below the level of the anterior corpora quadrigemina. In the center of this mass was an irregular shaped cavity filled with hyalin material. The tumor extended laterally almost to the left claustrum.

Prolongation of the tumor mass into the peduncle and pons, where it doubtless caught the fibers of the cortico-bulbar as well as the corticospinal tracts, probably explains the oculomotor symptoms. This corticobulbar tract, carefully worked out by Déjerine, conveys voluntary impulses from the cortex for associated movements of the eyes and head. In all probability interference with fibers of this tract at any point from the cortex to the cranial nuclei causes conjugate deviation of the eyes and head. In the patient such deviation was a transitory phenomenon marking a sudden exacerbation of the pressure, as is frequently seen in gliomatous tumors of the brain.

The emotional disturbance was probably due to involvement of the thalamic and subthalamic regions, as tumors of vascular disease of this region usually cause some disturbance of the muscular expression of emotion. The paralysis of upward movements of the eyes has been emphasized as a symptom of lesions at the upper end of the aqueduct of Sylvius, including movable tumors of the third ventricle. The symptoms of this nature in this case were doubtless referable to involvement of the region of the anterior corpora quadrigemina.

DISCUSSION

DR. HIRAM J. SMITH asked whether the paralyses of ocular muscles in this case were due to the involvement of the corticospinal tract or of the posterior longitudinal bundle.

DR. J. ELLIOTT ROYER thought the nature of the growth an interesting point. He pointed out that tuberculous growths are prone to affect that portion of the brain and have a tendency to be bilateral and grow rapidly.

DR. G. B. HASSIN said that tumors of the cerebral peduncles usually cause an ipsilateral third nerve paralysis associated with a contralateral hemiplegia, though a few cases have been reported in which a peduncular tumor did not give the typical Weber syndrome of hemiplegia alternans superior. In these cases the third nerve was not implicated.

He asked whether in Dr. Hamill's case the corpora quadrigemina were not involved. If so, the symptoms in his patient were due to a central or nuclear lesion of the third nerve. The proper diagnosis then would be a midbrain tumor with subsequent involvement of the large ganglions and of the cerebral peduncles, leaving the third nerve intact. Otherwise it would be difficult to understand how so large a tumor could spare the closely adjacent third nerve if it originated in the cerebral peduncles.

DR. RALPH C. HAMILL, replying to Dr. Smith, said the tumor occupied the ventral rather than posterior surface of the pons. It affected the fibers of the corticonuclear bundles. He could not say as yet whether the posterior longitudinal bundle was affected or not. The absence of the type of nystagmus

usually found in involvement of the longitudinal fibers made it impossible to say definitely whether this bundle was affected or not.

The history of the case emphasizing the early mental symptoms and the vomiting attacks, the beginning hemiplegia and absence of any true paralysis of the cranial nuclei, made it appear that it was rather an involvement of the corticonuclear fibers than of the cranial nuclei themselves.

The tumor was a glioma and extremely cellular. One of the sections showed typical breaking down of the tissue.

OSTEOMYELITIS OF THE SPINE WITH NEUROLOGICAL COMPLICATIONS (WITH LANTERN SLIDE DEMONSTRATION). DR. DALLAS B. PHEMISTER.

This paper will be published in a later issue of the ARCHIVES.

DISCUSSION

DR. PETER BASSOE said the second case reported by Dr. Phemister was of the greatest interest to him. He had never had a case like it before and considered it fortunate that they were able to make a diagnosis and have the patient operated on before the spinal canal was affected. When he called Dr. Phemister to see the patient, which he did at once, he saw that the condition was not meningitis. The striking thing about the case was the tremendous hypersensitiveness and the way the patient resisted all handling of the pelvis and the right leg; the septic fever with chills, the high temperature and high leukocyte count were also prominent features. Dr. Phemister was certain that they were dealing with osteomyelitis, but whether it was in the pelvis or spine could not be determined until the roentgenogram was taken.

Dr. Bassoe thought there were many cases of arthritis of the spine for which laparotomies had been performed, and in this case of osteomyelitis an able surgeon had operated for appendicitis. Recently Vanderhoof has reported a number of cases of spondylitis and arthritis of the spine with very misleading symptoms and resulting useless operations for supposed appendicitis or gallstones.

DR. A. B. YUDELSON asked in what percentage of cases of osteomyelitis elsewhere is osteomyelitis of the spine a complication later in life. Dr. Phemister had mentioned that some degree of ankylosis in the spine is due to myelitis. Dr. Yudelson wished to know whether the ankylosis was due to an actual myelitis, or a metastatic involvement from osteomyelitis in the extremities.

DR. D. B. PHEMISTER, replying to Dr. Yudelson, said he did not know how frequently osteomyelitis of the spine developed as a complication of osteomyelitis elsewhere, but in the case reported probably 60 per cent. had followed a focus elsewhere. The ankylosis of the spine that developed he believed was due more to involvement of the joints of the intervertebral spaces. Sometimes it would go beyond the bone primarily involved and affect three or four vertebrae, as does tuberculosis of the spine.

In the list of reported cases there are a few in which competent neurologists, most of them German, had diagnosed tumor of the spine, but on operation osteomyelitis was found. In one of these cases it was seen two years after the onset of the symptoms and the symptoms of infection had subsided, as they do in osteomyelitis elsewhere. The effect on the cord is frequently

lasting and permanent paralysis; marked paresis has resulted in some of the cases. Dr. Phemister thought that a good many of the cases of osteomyelitis of the spine have been mistaken for tuberculosis with cord symptoms.

PHILADELPHIA NEUROLOGICAL SOCIETY

Feb. 25, 1921

GEORGE WILSON, M.D., *President*

A NEGRO WITH HUNTINGTON'S CHOREA. Presented by DR. CHARLES W. BURR.

The patient was a man about 45 years old, of mixed blood, with a history of both gonorrhea and syphilis and a + + + + Wassermann reaction. His mother died after suffering with chorea for years. He was unable to give an account of his brothers and sisters. Mentally he showed the childishness of his race and in addition was somewhat demented. He received a gunshot wound in the forehead in 1918, but the chorea antedated the injury. He had marked characteristic movements. There were no signs of gross organic, spinal or brain disease. The interesting point was that of race. Dr. Burr knew of no careful statistics concerning the relative frequency of Huntington's chorea in negroes and Caucasians.

TWO CASES OF MULTIPLE SCLEROSIS. Presented by DR. CHARLES W. BURR.

The first patient was a man concerning whose condition there was a question of differential diagnosis between multiple sclerosis and paresis. He had scanning speech, nystagmus and intention tremor, and negative blood and spinal fluid Wassermann reactions, but the spinal fluid presented a paretic colloidal gold curve. When he first came to the hospital he was demented, but had no delusions. His apparent dementia rapidly disappeared, after which he showed no mental symptoms. A cursory glance at the literature reveals that at least one writer has found the paretic colloidal gold curve frequent in multiple sclerosis, even when there were no mental symptoms of paresis.

The second case was that of a man with multiple sclerosis in whom a partial monoplegia with local convulsions in the left arm followed the intraspinal injection of arsphenamin.

DISCUSSION

DR. ALFRED J. OSTHEIMER said that in connection with the difficulty in differentiating paresis or syphilitic meningo-myelitis from multiple sclerosis there recently have appeared statistics showing that the so-called paretic curve occurs almost without fail in the spinal fluid in multiple sclerosis, so that this factor is not one that can be used in the differential diagnosis. The Wassermann test, however, is a prime factor in the differential diagnosis.

DR. WILLIAM G. SPILLER said that the colored woman with Huntington's chorea, to whom Dr. Burr had referred, had been in his service many times at the Philadelphia General Hospital. She had the disease in a typical form with much mental deterioration. Dr. Spiller said he was not certain whether

Dr. Burr desired to emphasize the greater infrequency of Huntington's chorea in the colored race or not, but there did not seem to be sufficient evidence to support such a view.

DR. BURR said he had no statistics concerning the frequency of Huntington's chorea in the negro. In his personal experience it was rare. The woman at Blockley and the man he had just shown were the only two that he recalled having seen. There was no doubt that the negro had a genuine case of Huntington's chorea.

THE SIMULTANEOUS OCCURRENCE OF DYSTONIA LENTICULARIS IN TWINS. Presented by DR. GEORGE E. PRICE, Spokane, Wash.

The following patients were seen Nov. 14, 1919, in consultation with Dr. P. D. McCornack, who kindly placed their histories and his correspondence with their parents at Dr. Price's disposal.

Bobbie and Billie L., twins, aged 3 years, were born at 8 months, their mother being 19 years of age at the time. It was her first pregnancy. The twins were breast fed for six weeks. Both walked at 2 years and 2 months of age and talked at 2½ years. Teething occurred at the usual time. Both had convulsions which commenced three days after birth and lasted at intervals for several days. Bobbie had convulsions again when 6 weeks old. The mother stated that both babies had blue circles all over their bodies until they were 3 months old. About five weeks before they were seen by Dr. McCornack, both babies, during the same night, developed spasmodic, slow, cramplike movements of the entire musculature, except the face. These movements were most marked in the lower extremities. The muscular spasm disappeared after being present for several days, only to recur at intervals of a few days. Sometimes one twin would be affected alone, while at another time they would be affected simultaneously. During the spells there were loss of appetite and sleeplessness, and the children would be quite weak for some time after.

Family History.—The father was in good health. The mother had had attacks of momentary unconsciousness (*petit mal*) all her life. There was no other history of nervous or mental disease, nor of any condition approaching that with which the children were affected. There was one other child, aged 8 months, apparently normal in every respect.

Examination.—Both boys were well nourished, although they had lost considerable weight in the past five weeks. Both appeared to have deficient eye sight and one (Billie) had distinct strabismus (congenital). Both were mentally deficient. When examined, Billie was free from the spasm while Bobbie was affected. The contracted muscles were in tonic spasm, varying from time to time in intensity and alternating with periods of relaxation, causing a writhing or torsion of the extremities resembling athetoid movements. The tendon reflexes were normal in both cases, and there was no Babinski sign or ankle clonus. The lungs and heart were normal. Dr. F. G. Sprowl reported almost complete bilateral optic atrophy in both patients. The Wassermann reaction was negative. When tested with the galvanic current, the muscles contracted equally at either pole. This reaction occurs in myotonia. Erb's special reaction was absent.

Dr. Price's summary at that time was: An intermittent myotonia or dystonia associated with mental arrest and optic atrophy.

Subsequent History.—The subsequent history was interesting. In a letter the mother stated that on Nov. 18, 1919, while on their way home, Bobbie had an attack on the train. He vomited twice and shortly afterward became unconscious. Muscle contractions, at first mild, started in the face and extended over the entire body. After about an hour the motions ceased, and he lay motionless and apparently unconscious for some time. He then opened his eyes and tried to talk but was unable to do so. For the next few days he was very cross and nervous and took no solid food. In a subsequent letter, written July 19, 1920, the twins were said to be steadily improving, but no statement was made regarding the attacks. In the final report of Sept. 20, 1920, the mother wrote that Billie had had no attacks of the muscular spasm after leaving Spokane, and Bobbie had not had any since Dec. 18, 1919. She further stated that during any sick spell or digestive disturbance there was a tendency to convulsions. Their sight was improving so that they could distinguish objects close to them.

Diagnosis.—The conditions to be considered are hysteria, tetany, epidemic encephalitis and dystonia lenticularis. Hysteria at 3 years would not be impossible, but highly improbable, and could be definitely eliminated by the obvious illness of the children and the electrical reaction. The muscular spasm was not that of tetany. Trousseau's sign and Chvostek's symptom were absent, and the characteristic electrical reaction of tetany was not present. The absence of fever and the entire freedom from symptoms of any kind during the intervals between the attacks would be opposed to the diagnosis of epidemic encephalitis. Moreover, it would be difficult to picture a simultaneous infection with effects limited in each case to a small group of cerebral cells.

The muscular spasm alternating with relaxation simulating athetosis, the myotonic electrical reaction and the presence of marked evidence of arrest, point strongly toward a dystonia musculorum or dystonia lenticularis.

Is it not probable that the basal ganglions had to some extent participated in the general arrest or congenital defect, and that the cells of the putamen were dysfunctioning during the attacks of muscular spasm?

REPORT OF A CASE OF AGENESIS OF CRANIAL NERVES.

Presented by DR. WILLIAMS B. CADWALADER.

This patient presented a little known condition described by Möbius in 1892, under the title "Infantile Nuclear Atrophy."

The absence of function of the cranial nerves in this patient was noted shortly after birth and had persisted. Dr. Cadwalader believed it was due to defect of development of the cranial nerve cells; therefore he preferred using the term "agenesis" of cranial nerves, to distinguish it from atrophy, for atrophy would imply an acquired condition not truly congenital in origin.

History.—J. D., aged 10, was born at full term, the labor was normal and the patient was said to have appeared normal at birth. The mother stated that a few days later, however, the patient's eyes appeared to be "crossed," and she also had difficulty in sucking the bottle because of inability to use the lips properly. About this time it was also noticed that the patient was unable to close either eye completely, and later that the facial muscles could not be contracted normally. These conditions had persisted. In all other respects the child had been entirely normal, and was now robust and healthy. The family history was negative.

Examination.—Neither eyeball could be rotated outward; they both could be rotated upward and downward, but not symmetrically. The pupils reacted promptly to light and in accommodation. Both sides of the face were completely paralyzed. Though this condition was not recognized by the parents until some days after birth, Dr. Cadwalader believed it was present at birth, and he would attribute the bilateral absence of function in the muscles supplied by the sixth and seventh cranial nerves to a lack of development of their cells of origin in the brain stem.

DISCUSSION

DR. H. MAXWELL LANGDON said that there was no question that the patient presented had complete bilateral external rectus palsy. There was some weakness of inward rotation in each eye, although it was moderately well performed and better elicited when each eye was tried separately. The eyes were converged about 20 degrees when at rest, and any attempt at external rotation to either side caused overaction of the internal recti, increasing the convergence to 35 or 40 degrees. Upward rotation was well but unequally performed; the eyes in this action did not seem to coordinate. At times on upward rotation the right eye would take a higher level; suddenly it would drop, and the left eye would be the higher of the two.

Dr. Weisenberg asked whether she had binocular vision or fusion. Dr. Langdon said he could elicit no diplopia, but he did not know what the vision of the child was, and she might have such poor vision in either eye that in no circumstances would diplopia be produced. Dr. Langdon referred to another child, a girl 9 months old, with complete bilateral external rectus palsy, whom he had recently seen; in that respect this patient's condition was identical with that of Dr. Cadwalader's patient. The child was normal in every way except for the ocular condition. Birth was normal. During the seventh month of pregnancy the mother had had a violent attack of influenza. The fetal movements stopped for about ten days during the height of the illness. It seemed probable to Dr. Langdon that the child had influenzal toxemia which so affected the external rectus nuclei that they did not develop properly. He wondered whether some of the other cases of congenital palsy might not have been caused by some toxemia which the mother suffered during the pregnancy.

DR. SPILLER said that the pathology of nuclear aplasia had been inadequately studied. Möbius, especially, had written on the subject, and Siemerling had had a necropsy of one form of congenital ocular muscle defect (ptosis). The aplasia of the brain may be seen in other structures than the ocular or facial nerves. One of the most interesting forms is complete absence of the visual system, like that found in the case he reported in 1901, and which, so far as he knew, was the only case of the kind in literature. He had observed a young man who had no eyeball in either orbit. The optic nerves, chiasm and tracts were entirely wanting, and yet there was development, although not normal, of the nerves to the ocular muscles.

A PATIENT WITH A SECOND ATTACK OF FACIAL NERVE PARALYSIS: TREATMENT BY A SUPPORTING DEVICE. Presented by DR. N. S. YAWGER.

The case was that of a man aged 34 years, who ten years previously had had a right facial paralysis, at which time the routine treatment of internal medication, massage and electricity was given. The paralysis persisted for five months, and then a contracture developed. Following exposure, on

Jan. 2, 1921, he had another attack of facial paralysis on the opposite side. The patient came under observation two days later, when he presented an extraordinary appearance with his face hanging down heavily on the left and in addition drawn farther out of place by reason of the contracture on the right. Examination showed the paralysis to be complete with reactions of degeneration.

In this patient Dr. Yawger believed a supporting device would be of two-fold benefit—that the support in addition to holding the paralyzed cheek in place, would check the constant pull of the contracture from the opposite side. Dr. Yawger applied his device, and immediately a sense of comfort was afforded; later the patient said he could eat, drink and talk much more satisfactorily, and there was less exposure of the eyeball, which is often a source of great annoyance in these patients. The device was worn about a month, after which the cheek was almost able to sustain itself. The patient recovered within three months from the time of onset of the condition.

Dr. Yawger had not seen the patient in his first attack so that it was not possible to say that both attacks were of equal intensity. However, the second attack was well marked, and, occurring in the same person, it seemed to offer some opportunity for comparison as to the value of treatment with and without support. Recovery from the second attack, in which a support was used, occurred two months earlier than in the first attack; furthermore, the first had been followed by contracture.

Dr. Yawger had observed in this patient and in others that as motion was beginning to return, the implicated muscles became very sore, and he wondered whether this might have any bearing on the question as to whether the seventh nerve carries sensory fibers.

DISCUSSION

DR. CHARLES K. MILLS said that in a long experience he had seen a number of patients with facial paralysis who had recovered after four or five months, showing no or little improvement before that time. It was difficult to fix the time limit. As to time, he said there were three varieties of facial paralysis: one in which the patient recovered promptly after a week or two or a few weeks; one in which the patient recovered with moderate promptness, but comparatively slowly—usually in from four to six months; and the third class, in which recovery does not take place. Six months was a pretty fair limit for waiting. Dr. Mills thought Dr. Yawger made some remark about transmission of sensation in the seventh nerve and referred to a paper of Dr. Mills. A number of years ago Dr. Mills published a paper on "The Sensory Functions Attributed to the Seventh Nerve." His conclusions after many examinations and a large consideration of the subject and a study of Dr. Ramsay Hunt's papers, was that practically no sensation was transmitted in this nerve. Dr. Mills believed that Dr. Spiller thought that some sensation was transmitted by the nerve. Dr. Mills thought that the sensation supposed to be due to the facial nerve was really due to the fifth, this being extensively distributed closely in relation to the seventh nerve.

THE TREATMENT OF SPASTIC GAIT BY PERMANENT FLEXION OF THE TOES. Presented by DR. WILLIAM G. SPILLER.

Dr. Spiller presented a boy with great spasticity of the lower limbs. As spasticity of this kind can be overcome temporarily by bending the big toe downward, it had occurred to Dr. Spiller that by keeping the toes partially

flexed, as by a bandage or properly adjusted shoes, it might be possible to make a person with a spastic gait walk in a more normal manner.

The patient dragged his toes in walking. This gait was demonstrated. The feet were then bound with adhesive plaster with a small roller bandage under the toes, so as to keep the toes partially flexed. The result was striking. The boy immediately raised his knees abnormally high. By properly adjusting the degree of flexion of the toes it was possible to make the gait more nearly normal.

When the reflex of defense exists it can be produced voluntarily by flexing the big toe. This movement caused a contraction of the flexor muscles of the lower limbs. It is a well-known law, emphasized especially by Sherrington, that contraction of one group of muscles produces relaxation of the opposing group. By producing a persistent slight contraction of the flexor muscles by keeping the toes properly flexed, it appeared that the extreme spasticity of the lower limbs in extension could be overcome. This method of treatment needs further study. It is a question whether the reflex produced in this manner would become exhausted.

DISCUSSION

DR. C. K. MILLS, in regard to the remarks that Dr. Spiller made about the toe phenomena, wished to say that an old and valued member of this society, Dr. Wharton Sinkler, first pointed out the phenomena which resulted in certain cases from bending the great toe downward. Later some German neurologist believed he had discovered the same thing and published an article to this effect. As Dr. Sinkler described the matter, when the great toe was bent downward the foot was first flexed on the leg, then the leg on the thigh, and finally the thigh on the trunk. It was, in other words, what would now be called a defense reflex.

DR. SPILLER replied to Dr. Frazier that the Stoffel operation would be feasible, but it might not be necessary to do it. If the boy could be made to walk nearly normally by a simple contrivance, that might be better than operation.

A SERIES OF TUMORS OF BRAIN AND CORD SUCCESSFULLY REMOVED. Presented by DR. C. H. FRAZIER.

Dr. Frazier presented a series of tumors of the brain and cord successfully removed by operation in the neurosurgical clinic of the University Hospital. The tumors of the brain included:

1. A tumor quite unique because of its diminutive size, measuring in the largest diameter 2 by 3 cm. The tumor was accurately localized and proved on examination to be an endothelioma.
2. An endothelioma removed from the frontal region of a child 6 years old. The tumor measured 10 by 13.6 cm., and was interesting because of its huge dimensions and its gross resemblance to the brain cortex.
3. An encapsulated glioma. This was the first instance in the history of the clinic in which an encapsulated glioma had been found. The patient was admitted with a diagnosis of epilepsy.
4. A large endothelioma of the occipital region, accurately localized, removed in its entirety and without evidence of recurrence four years after the operation.
5. A tuberculoma removed from an adult negro. This was an interesting specimen because of its unusual size and because of the age of the patient

from whom it was removed. Tuberculomas are, as a rule, of small dimensions and almost invariably found in children. In this case the primary lesion was in the kidney.

Of the tumors of the spinal cord there were:

1. An extradural encapsulated endothelioma. The patient was paraplegic, and a diagnosis of transverse myelitis had been made elsewhere and operation not advised. The absence of pain throughout the course of the disease—two and a half years—was a conspicuous feature of the clinical history. Three weeks after the operation, sensation had returned, and the patient was able to walk.

2. An intradural endothelioma. Though present for four years, the patient was still ambulant. The diagnosis was based on the characteristic pain cycle, spasticity and moderate weakness of the lower extremities. The sensory phenomena were represented by pain and hyperesthesia.

3. An intradural endothelioma at the level of the first thoracic segment.

4. An extramedullary endothelioma removed from the cervical segments in a patient who had complete paralysis of both lower extremities and severe pain in the upper extremities. Though no larger than the tumor in Case 2 of this series, there was in one instance a complete paraplegia and in the other only moderate weakness of the lower extremities.

DISCUSSION

DR. FRANCIS X. DERCUM said that the small tumor which was removed from the patient with aphasia and spasm of the muscles of the face on the right side, was evidently partly subcortical. Furthermore, the aphasia was not persistent but only of transient occurrence and brief duration. It could have been due to interference with subcortical fibers. In other words, the speech phenomena may have been due to a diaschisis. The case can hardly be regarded as of localizing value for aphasia.

DR. CHARLES K. MILLS said he remembered one case he had with Dr. Martin in which a very small tumor in Broca's area produced the symptoms of aphasia. Another very small tumor was removed for him many years ago, by Dr. Keen, from the parietal region. In both cases referred to the growths were a little larger than the one reported by Dr. Frazier. The case of spinal tumor that Dr. Frazier referred to was a most interesting one. The woman had completely recovered; the tumor symptoms were not of a marked character, still they were decisive so far as the location of the growth was concerned. The woman had difficulty in walking and hyperesthesia and other symptoms, which led Dr. Mills to locate it where it was found by the surgeon.

NEW YORK NEUROLOGICAL SOCIETY

The Three Hundred and Eighty-Seventh Regular Meeting, March 1, 1921

FOSTER KENNEDY, M.D., *President, in the Chair*

A PATIENT WITH ACROMEGALIC FEATURES. Presented by DR. B. ONUF.

Dr. Onuf presented for diagnosis the case of a Jewish boy, 18 years of age, who showed some acromegalic features, but the picture was incomplete, and some manifestations did not tally with those of acromegaly. The hands and

feet appeared acromegalic, as confirmed also by radiographs. There was likewise a slight dorsal kyphosis, and the face was a little large. A distinct prognathism was, however, lacking, and there was no recession of the forehead and no thickening of the supra-orbital ridges. Most unusual, moreover, was the marked asymmetry of the upper extremities, the entire left one, and particularly the left hand, being markedly larger than its fellow and showing some increase of motor power. Another unusual feature, which could not be explained on an acromegalic basis, was a peculiar vascular or vasomotor condition. There was a mottling over a great part of the body and in some regions, particularly in the enlarged extremity, a succulence and increased vascularity of the skin. The mottling varied considerably under influence of temperature and other factors, being at times very marked so as to have first suggested a type of exanthema, at other times being just faintly visible.

Elephantiasis was thought of, but none of the authors who have studied this disease speak of any osseous enlargement of the parts affected; only the cutaneous and subcutaneous tissues are mentioned as the seat of the disease.

The patient alleged that the condition, referring particularly to the enlarged left upper extremity, was congenital, and his mother ascribed it to a maternal impression received while pregnant. While in that state she saw a woman with a very large upper extremity looking similar to that of the patient, and this sight affected her very much. In that connection Dr. Onuf mentioned another case seen by him casually in a business transaction. It concerned a man, about 40 years of age, a banker, who also had one unusually large upper extremity, which, however, showed nothing abnormal aside from its size, as compared with its fellow and the rest of the body, being otherwise well proportioned and of greater strength than its fellow. In this case also a history of maternal impression was given, the mother, while pregnant with the patient, allegedly having been much impressed with the sight of the Statue of Liberty with its one arm raised and holding the torch.

DISCUSSION

DR. W. C. BRUSH, in discussion, related a case similar to the one presented by Dr. Onuf, namely, that of a boy of 17 years, who was 7 feet tall. The disturbance was apparently of an endocrine nature and was familial, since one sister was a cretin and another had elephantiasis. In the whole family also the features were asymmetrical.

DR. L. PIERCE CLARK said that he was seeing similar cases associated with adolescence. Dr. Walter Timme had told him that it was not uncommon to have these patients improve spontaneously because in time an erosion of the sella allowed for a proper glandular activity of the pituitary gland, but as yet he himself had not been able to verify this teratologic adaptation. He said he would like to ask whether any other systemic symptoms, such as fainting or epileptoid attacks, had been observed.

DR. J. H. LEINER asked whether the blood pressure differed between the two extremities. Dr. Onuf replied that he did not think there was complete synchrony between the extremities.

BRAIN TUMOR OF THE MIDDLE FOSSA. DR. I. S. WECHSLER.

Dr. Wechsler reported a case of tumor of the middle fossa which was clinically parallel to a case presented at the last meeting. There was transient pain at first on the left side, then it became constant on the right. The first

neurologic examination was negative. The pain was not shooting or typical in any way and was thought to be psychoneurotic. The patient was not seen for two months, during which time the pain gradually diminished, on account of developing anesthesia of the fifth nerve. Meanwhile complete ptosis developed on the right side, gradually receded, and is slight at the present time. At the end of another month there was complete paralysis of the right external rectus. The pain had completely disappeared. The patient vomited once, but never saw double. At one time there was an herpetic eruption in the right corner of the mouth. The deep reflexes were normal, the right possibly a little livelier than the left. All forms of sensation had been lost in the right side of the face, the right side of the tongue and buccal mucous membrane. There were present, corneal anesthesia, keratitis neuroparalytica and beginning ulcer of the cornea. The disks showed no abnormality. Smell was not impaired. Weakness of the facial nerve was noted on the right; the eighth nerve was normal. Roentgen-ray examination of the skull showed normal sinuses; the right clinoid process could not be brought out clearly, there being apparently some erosion, otherwise, the skull was normal. Study at the Mount Sinai Hospital confirmed all the findings. Despite a negative Wassermann reaction, antisyphilitic treatment was given, but with no apparent benefit.

That the case is one of neoplasm of the middle fossa seems probable. The question, however, is whether the involvement is primarily of the fifth nerve, of the brain itself, or of the bone. The condition of Dr. Friedman's patient was very much like this, except that he had a disturbance of smell. Tumor of the bone in the case under consideration is perhaps excluded by the roentgen ray and the fact that the motor fifth is intact. The herpes would suggest involvement of the Gasserian ganglion. Some pressure on the sphenoidal fissure will account for the involvement of the sixth, third and the ophthalmic division of the fifth nerve. Possibly there is pressure on the cavernous sinus, which would account for the recession of the ptosis. The growth probably is not a sarcoma of the bone or glioma of the brain. An endothelioma of the Gasserian ganglion is most probable. A basilar meningitis is not likely to be limited to so small an area. The question of surgical interference is important.

DISCUSSION

DR. E. D. FRIEDMAN, in opening the discussion, said that the transitory character of ocular palsies was not uncommon. Serologic observations on the spinal fluid were lacking. Syphilitic meningitis, however, was rarely unilateral. Operative interference was not urgently indicated since there was no choked disk or other evidence of increased intracranial pressure.

DR. I. ABRAHAMSON believed we were dealing with a tumor, that the tumor was an endothelioma, rather flat and not encroaching to any extent on the cranial cavity; that in the absence of vital or more pathognomonic signs, an operation might be deferred. Complete removal of endotheliomas in that situation was very difficult.

DR. FOSTER KENNEDY asked whether the motor fifth nerve had been involved late or early. If late, it was improbable that the case was one of bone tumor; it was probably an endothelioma. If it came from bone an operation would be useless, but if it should be an endothelioma, a time would come when it could be operated on, and the waiting policy would deprive the man of any chance of recovery. Since he evidently showed no involvement of the bone, this would be the time to operate, if ever, or the growth would eventually kill him.

ANALYSIS OF A CONVERSION HYSTERIA SUPERIMPOSED ON AN OLD DIFFUSE CENTRAL NERVOUS SYSTEM LESION.

DR. PHILIP R. LEHRMAN.

Dr. Lehrman pointed out the difficulty of detecting symptoms of hysteria when associated with organic neurologic signs. This combination not seldom appears to be a definite syndrome of an organic nervous disorder. An illustrative case was that of a young woman 23 years old, who came to Vanderbilt Clinic for advice concerning a coarse, irregular, intention tremor of both hands and fingers, which had begun at the age of 12 years, and had progressed unfavorably despite treatment for the past eight years. At one hospital a foot deformity was operated on, and the condition was diagnosed as pronated feet and Friedreich's ataxia. She was almost entirely incapacitated by the tremor. She complained of a great deal of pain of a deep, burrowing character, starting in the base of each thumb and traveling up the forearms to her elbows.

History.—At 2½ years of age she fell off a high chair, following which she stated that she had a left hemiplegia and aphasia which soon improved. Until 12 years of age she dragged her left foot and showed weakness of her right hand. This was regarded by some physicians as residuals of poliomyelitis. Her father and mother were short of stature. The maternal uncle showed tremor of the hands when writing. One sister's deep reflexes were absent. There were no other mental or nervous diseases in both ancestral branches.

Physical Examination.—The positive findings were: height, 4 feet 5 inches; pes cavus; hyperextended fingers at knuckles; toes plantar flexed. Gait showed slight dragging of the left foot. She was unsteady in the Romberg posture. Nonequilibratory tests (f: f, f: n) were badly performed on account of tremor. Dysmetria was present on the right. All deep reflexes were absent. The Babinski reflex was questionable on the right. Muscle strength was absent in the toes, limited in dorsal flexion of the feet, especially on the left, and limited in the upper extremities, more on the left. The Grasset-Bychowski reflex was present. Sensation to touch was diminished in the toes and fingers; the vibratory reaction was diminished in the lower extremities. The right pupil was larger than the left. She reacted peculiarly when a vibrating tuning fork was placed near her ears; the sound seemed to startle her and she would tremble. All other findings were negative.

Mental Examination.—Exploration of the unconscious was resorted to to find evidence of a definite mechanism. The analysis, January to May, 1920, covered a period of about thirty-seven hours of interview. The problems to be solved were: Why did she develop neurosis? Why were these particular symptoms caused, and what was their meaning in terms of the unconscious? The patient's account of her early life in Russia and in America indicated mistreatment by her parents. The father was said to have deserted his family; the mother, however, followed him to America and a reconciliation took place. The patient always hated her father, and he reciprocated this antagonism. She felt constantly abused, thought she might be an illegitimate child, and on the birth of a sister when she was 11 years old, who became the center of the affections of the parents, the patient felt that her surmises were true. Other brothers and sisters had died of neglect. She herself had been injured as a child, purposely, she felt. When the little sister was about 10 months old the patient accidentally dropped her down the stairs. This frightened her for she knew that she would be punished. Her whole body trembled with

fear. The next day she was unable to write at school because of the trembling of her hands. This was the beginning of the tremor.

Her increasingly intolerable position in the home led to day dreaming of Cinderella-like situations in which she would finally be vindicated and taken to a pleasant home by some rich old gentleman. The major part of her narration suggested in tone inflection and phraseology a complaining 12-year old child. From an emotional child, however, she would at times change with striking rapidity into a sneering, ill mannered adult, and would assume an emotional stupidity, a sort of Ganser syndrome on the emotional level. In most respects she lived as a child of 12.

All her day dreams were of the same fabric, that of a suffering personality. It was therefore reasonable to suspect that her view of her life was distorted, and her tale spun of a material the underlying basis of which was the predilection for situations of suffering. The question suggested itself: If she could invent phantasies in which she suffered, why could she not have invented real situations in her home in which she could suffer? When the events of her life were retraced in this more critical light, an entirely different story was apparent. The patient's trouble was not alone the tremor of her hands, but what perhaps was more important, her distorted view of life. It was necessary to correct the latter, to make her view critically her inadequate reactions to certain situations of early childhood and adolescence, and not until that was accomplished did she gain sufficient insight into the motives in the productions of her symptoms and their final abandonment. Because the beginning of the tremor had coincided with an intolerable situation at home where she was supposedly abused, it did not necessarily follow that the alleged abuse caused the tremor. Instead there was a common basis in the patient's unconscious for the alleged abuse (which was not a cause but a symptom of the neurosis) and the resulting tremor. The forces at work were an accentuation of the sadomasochistic component with a masturbation and a prostitution conflict. The analysis indicated the manner in which these forces began to exert their pathologic effect. The patient was handicapped early in life by being crippled. Since her father had emigrated to America she had had a "thousand fathers and a thousand mothers," relatives and village neighbors, and received more than her share of pity. Circumstances prevented her mother and father from giving her the normal amount of love. The love she received was from strangers who pitied her, and she sensed early that it was her suffering which made them do it. This started her "career" of suffering. Finally a number of events precipitated the tremor. At 11 years she began to menstruate and had masturbatory experiences. About that time also her infant sister consumed all the attention of her parents. Jealousy was aroused and the "accidental" fall of her sister followed. The tremor developed the next day. The function of the tremor, therefore, was to prevent her from doing things which she unconsciously desired. In dreams, her hands always performed the same function of protection.

As the patient gained insight into her unconscious motives, the tremor gradually began to disappear until she was able to manicure her nails and thread a needle. Conditions at home she said were becoming much more pleasant. She realized that it was she who had changed and not her environment. For the first time in years she was on speaking terms with her father.

DISCUSSION

DR. L. CASAMAJOR reported that at the Vanderbilt clinic the case was considered one of mal development of the central nervous system. The choreo-

athetotic movements led to the belief in an organic cause for the condition. The patient's violent fright reaction to the tuning fork gave the first suspicion of hysteria. Only after the analysis did the tremor disappear. When the patient was last seen, the organic features, foot deformity, etc., still remained, but the psychic features had disappeared. The patient was extremely short in stature, and this with the organic handicap gave a basis for psychic compensation. After the removal of the neurotic elements, the primary organic disorders still remained.

DR. STERN said that analysts could realize the great difficulty in putting into a short paper, the many months' work which the analysis of a case entails. He also said that the period of thirty-eight hours, which was the time given to the patient by Dr. Lehrman, was a remarkably short period of time for such good results. Another interesting feature was the association of a functional with an organic condition, necessitating careful weighing of the symptoms present; in this connection it may be mentioned that the organic condition is not necessarily a causative factor, for similar psychic states exist in patients not so handicapped. The important factor is the psychic make-up of the person and his way of reacting to certain situations. Dr. Lehrman's paper also gives us a glimpse into the causes of human behavior, indicating, at least in neurotics, behavior in adult life having factors that should be traced back to infancy for proper understanding of that person's adult behavior.

DR. JOHN T. MACCURDY said that Dr. Lehrman deserved hearty congratulation for the success of his analysis. Those who have ever attempted psychoanalytic work in dispensary practice are well aware of the extreme difficulties attending this procedure. Dr. Lehrman's paper was further interesting in that it presented the problem of diagnosis in cases in which both organic and functional symptoms existed. Dr. MacCurdy wished to express his opinion that such diagnoses are much more easily made if the view is held that functional symptoms are the product of definite etiologic factors, just as are symptoms with an organic basis. In other words, a diagnosis of psychoneurosis should be made when a faulty make-up is demonstrable and the precipitating cause determined, which is of a kind that precipitates neurotic reactions. Without such data the condition is probably purely organic, but if these factors are present, the condition is either purely functional or has a large functional element. In this connection Dr. MacCurdy mentioned a survey that had recently been made of 100 consecutive admissions to the Medical Dispensary at Cornell. Each patient was first questioned by a psychopathologist who made a diagnosis of the organic or functional condition on the basis of the mental attitude and history of the patient. This meant a positive diagnosis of functional disease and a diagnosis of organic disease by exclusion. Each patient was then examined thoroughly by internists who made organic diagnoses on a positive basis and functional diagnoses by exclusion. In correlating the results there was a difference of opinion in only six cases, and 42 per cent. of the cases were found to be purely functional. Dr. MacCurdy expressed the opinion that an adoption of the method of positive diagnosis of the functional condition, rather than diagnosis by exclusion, would greatly increase the accuracy of our diagnoses in general practice.

PROGRAM FOR THE STUDY OF HUMAN BEHAVIOR. DR. STEWART PATON.

Dr. Paton of Princeton University (by invitation) outlined his program of action for treating some of the human problems of the day. A more prominent part must be taken by the medical man in public affairs now, and a

definite program is still lacking. The great human problem in the world at present is that of human behavior. Dr. Paton's program would include a study of this. During the war psychiatrists were asked to help the government determine the predisposition of drafted persons, both in army and air service, for warfare. Those whose systems would probably crack during the strain were weeded out and not sent for overseas duty. The leaders of men could be recognized and used where they would be most efficient. There is just as great a field for neurologists and psychiatrists to pass on the predisposition of persons to be leaders. If it is believed that leaders already in power are not effective, an effort should be made to educate the public to this point of view. Moreover, the selection of trained men for industry, for negotiating labor problems, is an all important question. Labor problems often owe their existence only to the predisposition of the opponents and their consequent misunderstanding. In the present crisis abroad, only the arguments in favor of peace are considered while the predispositions favorable for peace are not taken into account. The wishful thinker, superidealist, is doing a great deal of harm in the world today, and is allowed to act without restraint. It is time for the world to emphasize the importance of the study of the human personality. Public men must be imbued with this idea. The financial end of establishing departments for the study of human behavior in medical schools is a stumbling block; yet other departments in universities receive endowments and special research funds. The astronomers have formulated their problems and are able to show what they want and need. The business man appreciates definiteness in detail, and his money and interests go to further work in which the end is known. The tendency of the program for the study of man is to overemphasize the importance of the analytic side, that is, the study of different organs and parts of the machine. A synthetical side, the study of the reaction of the individual as a whole, has been neglected, and this should be developed.

A pure research center for psychiatry situated in New York is urgently needed. New York should have the greatest center of this kind in the world since she has the greatest need for it. Provision should be made in this research center for training neurologists and psychiatrists, as well as those interested in educational, social and ethical problems. Here human behavior could be observed and checked up by clinical study. It is not necessary, however, to wait for the research clinic. The practitioner interested in the analytic side of his work should widen his interest so as to include the synthetic side and observe his patients as living beings adjusting to meet actual situations in daily life. This kind of study can be carried on to great advantage in general medical dispensaries. The opportunities for psychiatrists here would be limitless, but the difficulty will always be, until special training centers are founded, to get men properly equipped to fill the positions.

A new and untouched field for the study of human behavior is opening up in the university in which there is no medical school. The average college student is trying to find himself emotionally and mentally. A sizing up process is constantly active—problems of the individual in relation to religion, sex, attitude toward the world and the world's attitude toward him. Several types of students may be readily picked out: the so-called normal student, who makes an easy adjustment; the inadequate student, who assumes an imaginary importance to the university, based on his record in sports or studies. This type never comes quite up to the mark in spite of swagger and bluff, and is constantly trying to adjust himself. Some of these students pass through the

university and others drop out. Then there is the "sorehead" who, because of his failure to adjust himself, feels that he is singled out for special injustice. This injured, defensive attitude in such individuals unfortunately lasts through most of their lives. The attempt to correct these defects should be made before the university period, in early school days. Another interesting type is that of the boy with the original mind. This type, as a rule, is not fully appreciated. His intelligent curiosity and genuine interest in life does not lead him to books, nor does it develop the qualities necessary for passing academic tests. Gradually his inquisitiveness is killed, and he becomes intellectually indifferent. He has little chance of passing through the university, if he refuses to conform to convention. A great deal can be done for this type, and should be done. The world should be educated to the point of giving such a boy a better chance to develop his natural endowment.

DISCUSSION

DR. E. J. KEMPF said that he believed in the material presented by Dr. Paton. He was particularly interested in the psychopathic person in high school, in college and in business. Study of such characters will have to be carried on on an individual basis. The student of normal and abnormal behavior must try to analyze his subject's difficulties and help to synthesize a career for him which would gratify his unconscious, as well as conscious, cravings. The choice of a profession in business men is motivated by unconscious repressed wishes; these design his friendships, his enmities, his failures for him. His whole business life consists in an effort to work out his unconscious cravings, and he tends to remain blind to his own defects. Here is an enormous field for study and interpretation.

DR. GREGORY STRAGNELL (by invitation) expressed his interest in the presentation. The approach to these problems by the educator is unlimited when he understands what he can do. He has the opportunity of taking up the problems where the parents have failed. Education, even in the primary classes, should encourage the child to give expression to his personality instead of putting him through a machine-like process. A training institution would have scope for work at both ends, and educate the educator as well as the child. So many of the youths in Dr. Paton's third class fail simply because the educator feels that he must keep down to certain levels, where he can retain his dependence on books.

DR. L. PIERCE CLARK said that the question of predisposition for business success or any form of success is one of development of mind and character. With some it is a natural gift. Personality has much to do with it. The fundamental thing for the present seems to be the restatement of the development of the successful life which may be presented to adolescents.

DR. I. STRAUSS said that any hope of reaching a solution is lost if we have to define a successful life. It is nothing new to hear a professor say that the college does not educate. The problem in Dr. Strauss' opinion must first be attacked analytically. It is too early to make the synthetic attack. When education is to be attacked we must begin with the parents.

The exceptional boy has no outlet, and the educator could not make an adjustment for this individual. Something might be achieved if these types could be segregated and specially instructed. The seal of approval, however, is the desired goal of most men. One good move has been made by the founding of a clinic in which the exceptional boy can be studied and analyzed, and a prescription for future conduct issued. This process is, however, a very expensive one.

Where are men to be educated? As soon as research work shows the need of such special training the work will begin. The medical school does not pay enough attention to behavior, it is true. The successful practitioner is the man who by reason of his personality can handle human beings as such. The medical schools should aim at this individualistic concept. Courses on human behavior which might be developed would not find acceptance in the medical schools, since the need has not yet become apparent, and there is no interest outside of actual technic. The tendency has been for the medical profession to shut itself off and remain quiescent on a high pinnacle. Until we can come down to the level of the rest of the community, nothing can be done. The same apathy has been striking whenever questions of public cooperation have been raised, the Workmen's Compensation Act for instance. Advances have already been made in the study of the personality in connection with employment in business, but the medical profession is doing nothing to direct this work. First must come research, then propaganda, then after showing the need for this type of work, we can begin to plan a program.

DR. B. ROSENBLUTH told of his experience with people, mostly of the working class, whom he has given the facility to come in to see him at stated intervals. The people's great want, experience with this class shows, is the desire for discussion of their problems so that they can make their own conclusions from the premises of the discussion. The sense of loneliness of the individual is the most striking feature. This results from the necessity of each person working for self. The army experience is valuable because self is eliminated, and the value of the individual is estimated for communal good; that is, a man must be a factor in relationship to the safety and efficiency of the whole unit, and not in relation to himself. This experience should be applied to civil life, and the individual should be taught that existence is not for self only but in relationship to others.

The strength of the individual is strength only as far as it is the strength of the social unit to which he belongs. Hence education should be directed to adjust the individual automatically to those about him, and not merely to adjust himself into some part of the community in which he finds himself comfortable.

The failure and "sorehead" is the person who sees only himself, and feels that nobody can appreciate the qualities which he knows he has.

DR. FOSTER KENNEDY pointed out that the Roman Catholic church asked for the education of the child up to the age of 7 years, realizing the importance of the early education. Physicians as a rule do not read enough history. If they did, they would notice that those nations in the past have stood highest in which potestas patris was strongest. With the decline of control in the family, the nation as a whole declines. Rome, China and the Jewish race show this. The maintenance of the family unit is the integral factor in the maintenance of the herd. The exceptional boy will take care of himself eventually, but it is the mass that must be educated. The problem of education will be greatly simplified when children are reared properly in the nursery.

DR. PATON, in closing, said that he believed there were reasons justifying optimism in regard to carrying out the program. The deans of colleges were anxious to cooperate and were greatly interested. If the medical profession could supply the men—those who understood and could interpret human behavior—the services of these experts would be sought. The recognition of the need for men trained in the subject of human behavior was developing faster than the supply of experts. The fate of democracy depends on the measures taken to supply the demand.

Book Review

PSYCHOPATHOLOGY. BY EDWARD J. KEMPF, M.D., Clinical Psychiatrist to St. Elizabeth's Hospital (Formerly Government Hospital for the Insane), Washington, D. C.; Author of "The Autonomic Functions and the Personality." Pp. 762. St. Louis: The C. V. Mosby Company. Copyright, 1920.

In his first book, "The Autonomic Functions and the Personality," Kempf evidenced his rich psychobiologic imagination, which made us await with keen interest the present work. It promised to bring concrete material in which the conception of special autonomic strivings and their conflicts could be studied. Perhaps the most insistent and consistent of our younger American psychopathologists, a man with a theory and a tremendous determination to push it to its logical and practical end, Kempf has indeed brought himself to a fuller expression than any present-day worker in this field.

With this volume Kempf has earned the credit of giving the first consistent account of a wide range of mental disorders viewed from the angle of sex-pathology and the struggle for what he formulates as virility, goodness and happiness. Ninety-three well-written case reports form a highly commendable record of less than a decade of well focused work at the Indiana State Hospital at Indianapolis, the Henry Phipps Clinic and the Government Hospital of Washington, D. C. Viewed from this angle, the material offered is, to the reviewer's knowledge, the most noteworthy product from any single worker and, he does not hesitate to say, from any aggregate of workers. It is a unique record—and would be unqualifiedly the most remarkable production of our period if it showed evidence of a safer scientific basis.

The series consists of three "anxiety neuroses," seven "psychoneuroses" (hysteria and compulsion states), thirteen "manic-depressives," five "paranoics," thirty-six cases of paranoid dissociations, nine cases of catatonic dissociations, seventeen cases of hebephrenic dissociations, two cases of general paresis, and one of arteriosclerotic deterioration.

These cases are discussed under these interesting headings: Suppression or Anxiety Neuroses (pages 201-288); Repression or Psychoneuroses, their mechanisms and relation to psychoses due to repressed autonomic cravings (pages 289-252); Benign Compensation or Regression Neuroses, with or without dissociation of personality, manic-depressive psychoses, elimination or simulation of wish-fulfilment in affective crises (manic-depressive psychoses, pages 353-420); Pernicious Repressive Compensative Neuroses, the psychopathology of paranoia (pages 421-476); the specially important Chapter X, the psychopathology of the acute homosexual panic, acute pernicious dissociation neuroses (pages 477-515); the psychopathology of chronic pernicious dissociation of the personality, with defensive hatred, eccentric paranoid compensations and pernicious deterioration (chronic paranoid dissociation, pages 516-555); the psychopathology of chronic pernicious dissociation of the personality, with crucifixion and catatonic adaptations to the repressed cravings (chronic catatonic dissociation, pages 556-614); and the psychopathology of chronic perni-

cious dissociation of the personality, with hebephrenic adaptations; predominance of excretory erotic interests (chronic hebephrenic dissociation, pages 615-697).

The setting is furnished by the following chapters:

Introduction (pages 1-19); physiological foundations of personality (pages 20-75—in many ways a restatement of the essence of his first book); the psychology of the family (pages 76-117); the universal struggle for virility, goodness and happiness (pages 118-178); the influence of organic and functional infirmities on the personality (pages 179-188); and the mechanistic classification of neuroses and psychoses produced by distortion of autonomic affective functions (pages 189-200). The casuistic chapters are followed by: A reconsideration of the conditioned and repressed autonomic affective determinants of abnormal behavior (pages 698-732), and a brief chapter on "psychotherapeutic principles" (pages 733-751). Two pages of bibliography and an index close the book.

The whole presents a rather formidable task for the reader and reviewer, but one which is likely to hold the attention, especially in the concrete material.

An attempt to restate the work at once confronts one with the intensely personal spirit of the whole book. Kempf is a man of one dominant idea and principle, that of the "autonomic strivings" for gratification of the sex instinct and of the struggle for success in winning both the goal and the esteem of the object of adoration. The collection of case records which he presents as a basis for his "Psychopathology" and discussion of principles is a remarkable display of ever recurrent motives and problems of this type, each record well presented and clearly focused, although obviously with a preeminently sex-dynamic vision, comparable to a study of the visual world made only with ultra-violet rays. In this and in the plastic and directly and frankly demonstrative method of illustrating his ideas with no less than fifty illustrations from pictures and statues of ancient and modern art, in addition to pictures of, and by, patients, Kempf has furnished the bald explanation of a great deal that was left in uncertain outlines in his previous book.

It may be most advantageous, after the summary survey, to begin with Kempf's series of case records, to review them for the facts and suggestions they offer, and then to review the theoretical deductions.

A number of cases can be grasped simply and directly in terms of any kind of pragmatic psychology, irrespective of the proposed theories.

In many of the case records, Kempf leads one to assume a positive "segmental autonomic striving," wherever any symptoms occur that would refer to the anal, oral, balanic or vaginal zone; any fancy or hallucination, whether with the positive, aggressive or defensive sign, must denote this "autonomic striving." In order to keep up the tremendous pressure under which his formulas must hold and work, he is as unable to tolerate a middle ground as is Freud with his obligatory psychodynamic determination of *all* the memory lapses and the like. The idea that some of the reaction-tendencies *might* be swung in incidentally and be dangerous only because of the biologic ease with which they can *become* dominant, would probably be intolerable to him. It would take away the obligatory validity of his neurophysiologizing scheme, which, we think, can figure only as a subsidiary hypothesis. One could be much more sympathetic toward a conception which would speak of *relatively* "autonomous" (not purely "autonomic") reaction-tendencies, more or less characteristic of the eccentrically reacting individual or family, or charac-

teristic of the type of break of compensation. But this would render negative the chosen scheme which makes of the living being a series of segmental autonomic organs of appetites, sticking out sensory surfaces so as to be sure to catch all the contacts and agreeable tickles needed for the gratification and neutralization of the cravings and incidentally the satisfaction of the biologic needs.

In this continual reference to the autonomic segment and striving, undoubtedly one is better guided to something more concrete and biologically helpful than with old-fashioned nerve-cell talk. But why replace it by an autistic nerve-physiology? The facts could be just as adequately and correctly expressed in common-sense psychobiologic terms and situations, unless we should really be able to single out specific neurophysiologic patterns accessible in neurophysiologic terms and experiments, working and workable in a simple physiologic manner.

Kempf yields to the ingrained psychiatric obsession to offer a "*classification of neuroses and psychoses*." It is a "mechanistic" *classification*, in contrast to a nosologic one, but also in contrast to the freer and more plastic dynamic *formulations*. It is mechanistic in showing how a "distortion of autonomic-affective functions" produces definite types of "neuroses" (and psychoses) characterized according to the "mechanism" as suppression-neuroses, repression-neuroses, compensation-neuroses, regression-neuroses, and dissociation-neuroses. He specifies in his table (reproduced in the *Journal of Nervous and Mental Diseases* 50:105 (Aug.) 1919, and in Jelliffe and White's "Nervous and Mental Diseases," Edition 3, p. 784): the diagnostic classification; the grouping as benign and pernicious; the mechanistic differences of the five types mentioned in the foregoing; the common symptoms; the common causes and the old diagnostic terms. *Suppression neuroses* go with clear to vague consciousness of the nature and effect of the ungratifiable affective cravings; *repression neuroses* with "vague consciousness to total unconsciousness of the nature and influence of the ungratifiable affective cravings"; *compensation neuroses* with "persistent striving to develop potent functions and win social esteem initiated by fear of impotence or loss of control of asocial cravings"; *regression neuroses* with "failure to compensate but regression to a preceding more comfortable, irresponsible level, permitting wish-fulfilling fancies, postures and indulgences"; and *dissociation neuroses* with "domination of the personality by the uncontrollable cravings despite the efforts of the ego to prevent it." The enumeration of the "common symptoms" shows the difficulty of carrying through these differentiations which are by no means always exclusive of each other and therefore should figure as *principles* to be considered in the free formulations of the cases rather than as a "classification" of the "neuroses" and psychoses, and much less as a classification of *case*, a hankering excusable in a naive statistician but not in a modern clinician. Kempf gives the double-edged advice: "We must not forget that the golden rule in diagnosis is to know what we are looking for because then it is infinitely easier to find it" (in italics). He bows to the traditional use of the descriptive terms "acute" (of less) and chronic (of more than a year's duration) and periodic (for periodic, intermittent or recurrent). The "autonomic-affective conflict" is benign or pernicious according to whether the ego can accept it or not and when it is of a postadolescent or infantile type. Cases can change their classifications; a pernicious typical "paranoid dementia praecox," on the development of a transference to Kempf, could be brought back to a simple benign suppression-neurosis.

One serious drawback in this scheme is the difficulty of obtaining a generally acceptable and fixed meaning of the terms used. Rivers uses exactly the opposite meaning in the terms repression and suppression; the term dissociation is generally used for a broader conception than Kempf's, including the benign as well as the pernicious dissociations; moreover, in actual work one finds that the various "mechanisms" can coexist and do not exclude each other and have by no means always the same benign or pernicious meaning.

A psychophobia unnecessary in this age of explicit or tacit acceptance of the concept of psychobiologic integration comes out in the paragraph on page 199: "In conclusion, the term 'psychosis' is not used because, after all, the sensory phenomena which we are conscious of as thoughts and wishes are really integrative physiological processes and the term 'neurosis' is more consistent with the integrative functions of the nervous system." Kempf has not got beyond the physiologizing integrative functions of the nervous system, and yet he works altogether with integrations only fully intelligible in psychobiologic terms. The positive side of the scheme is nevertheless of great stimulating and clarifying value, if given as a scheme to give *a dynamic formulation of the facts in a patient*, and it can properly be carried out even when one has cause for doubt concerning the exclusively autonomic character of the affective and other dynamic factors.

When one goes through the cases one certainly meets a wide range of facts which are practically all expressed in terms of psychobiologic and psychodynamic factors and reactions commonly accepted and understood, and relatively few which could be said to be demonstrable as of intrinsically and purely autonomic origin. The case records have their force in the psychodynamic approach and much less clearly in any demonstrated merit of the theory. One is continually reminded of a kind of contrast of the "ego" as the supposedly good or at least acceptable element, and the autonomic craving as the "flesh" of a not especially biologic type of ethics—no doubt a simple and practical scheme but one playing into the hands of those who may be too quickly and too dogmatically influenced when their turn will come to hold a fixed scheme over the patients and over the world. Kempf's own moral philosophy seems to be sufficiently fluid to satisfy even the most radical yearnings for sex-emancipation; but the revulsion which may come when the present generation assumes authority over the next generation may make one yearn for a less cut and dried scheme than that of the conflict of ego and flesh.

For a quick review of the conception of man and human problems as Kempf sees them, a study of the illustrations and their legends furnishes a vivid summary such as, to the reviewer's knowledge, no other author has given us. We find there the serpent and phallus and the bowl and the vase, the signs of union, the incest problem, the popularizing and refining of sexual interests, "insuring the race against autoeroticism, prudishness, prostitution, perverseness and suicide;" the anguish and regret at sexual waste, the agony of uncontrollable, ungratifiable sexual cravings, contrasted with the picture of "contentment of a normal biological career," the association of nutritional and sexual interests; the struggle against homosexuality, the regression of masculine virility to effeminacy after slaying a friend; the eternal vigor and constructive power of uncensured heterosexual love; the agony of woman's mission of maintaining the earth and fruits thereof; the vase of the caryatid as the burdensome uterus and longing for maternity; the storm "a love fantasy pursued by censorship;" the ring "an uncensured love fantasy;" the Madonna of the Rose, with traces of longing in the features and a costume indicating a highly sublimated attach-

ment to the father; the mother and child worship; Bacchante, a modern version of joyous motherhood, freed from religious suppression and dogmatic conventions; the "requiem," and Boecklin's Toteninsel, depicting the affective significance of suicide as return to the uterus; Buddha the sublimation of autoerotic selfsufficiency; types of copulation, stimulation of reproduction; fancies of impregnation; Graziella planning to catch the lover with the net; longing for maternity; Eve in anguish following the censored sexual act, etc. Postures and productions of patients are further illustrated by archaeology and classical and modern art, to bring out the struggle against and flight from the autoerotic, the analerotic, oral-erotic and incestuous "segmental strivings." Stuck's sphinx (persistently called *der sphinx*) shows a young man destroyed by the selfish, bestial, incestuous love of the mother. In short, Kempf gives a remarkable array of pictures with many ingenious interpretations partly suggested by patients and a very clear and helpful illustration of the author's vision of art and artistic imagination.

The same sagacity and directness hold in Kempf's reading of the problems of the family and the history and fate of various combinations of temperaments. The willingness to state as definite generalizations many a fact derived from the histories of cases goes with the keenness of Kempf's capacity to use all experience in concrete problems of family and human relationships as part of his psychiatric world of thought. It is this capacity to see and to use life that brings him close to what is meant by the "common-sense" which he belittles in others when applied to the problem of consciousness and the philosophical and logical foundations of psychopathology.

Kempf is one of those who found in Freud's hysteria studies and the concept of freudian analysis a tremendous help for liberating their own dramatization of human problems and those seen in patients and in art and in a general conception of human life. For any one who wishes for an epitome of Kempf's standpoint, nothing could be better than to read one of his earliest analyses (P. N. 1, page 293, also published in the *Journal of Abnormal Psychology*, [April, 1917]) and his review of the "Yellow Jacket" (*Psychoanalytic Review* 4:393-423), unfortunately not taken into the book in toto, but, at least in the original, in the description of childhood and adolescence, the best thing Kempf has ever written.

Like Crile, a precursor of Kempf in the appreciation of emotions in medicine, Kempf seems to have found in Darwin his somewhat belated liberator from a dogmatic bringing up. The unrelenting "struggle for survival," the power of the emotions and the compelling rôle of limited visceral mechanisms in the activation of the emotions have led Kempf to the somewhat grotesque but nevertheless illuminating conception of the relation of autonomic and "projicient" mechanism of man. Freud's panerotization of dynamic psychology and Sherrington's concept of a final common path and postural tensions, are combined with Kempf's own tremendously keen desire to shape life and to give it a fervor and artistic fulfillment and conquest in the struggle for esteem. A need of feeling and of expressing his vision of human nature and human facts in terms of emotional superlatives is illustrated in his style and choice of terminology. With it goes also a staunch resistance to anything that would moderate the contrasts with which the picture as he sees it could alone be staged to full advantage. Fervor, strenuosity and an attitude of the bitter-ender mark the method and the result; and the unwillingness or inability of reintegrating the psychology as taught him by his early training is the main limitation of the scheme and the cause for far-reaching overcompensations.

Kempf has to speak and think in terms which represent situations of great affective meaning to him. He sees and uses analogies which the average physician would hardly be tempted to apply. Passivity becomes crucifixion, restraint castration, etc.

With the emotional appeal, there goes, however, a decided lack of concern about the scientific and critical solidity of the speculative ground on which he chooses to build. On a more modest basis his formulation of man as a self-constructive and to some extent self-steering product of integration would be acceptable and will coincide with other concepts of integration. But on the one hand he falls short of it, and on the other he makes assumptions which few serious workers would be willing to endorse; and the whole becomes a narrowing scheme, fortunately richly compensated for by a wealth of data of observation and conjecture which makes one condone the relatively extraneous theoretical claims.

Kempf overcorrects the intellectualizing tendency of tradition by creating a supremacy of autonomic segments and their strivings, segments which "create" and maintain the "projicient mechanisms," i. e., the central nervous system and the somatic as opposed to the visceral body.

Kempf is so fascinated by his formulation of the autonomic segments and the procurer's rôle of the projicient nervous system that the adverse results of Cobb's experiments (*American Journal of Physiology* 46:478) could not affect his scheme. Sherrington's experiment on the emotions is not mentioned, whereas it might well be remembered as a balance to the too one-sidedly exploited studies about the final common path and the postural tensions. One is reminded of Robinson's continual pleas for the "abdominal brain" and of Pfaff's criticism of the oral digestion theory of Fletcher, which he doubted because if Fletcher were right, the vertebrate would probably have a mouth several feet long and a much shorter intestinal tract.

Just as the old phylogenetic recapitulation theory so long exploited by Stanley Hall, Kempf's combination of freudian technic, Thomistic philosophy and Sherrington's integrative functions of the nervous system yielding the contrast of the autonomic and projicient nervous system will help some students on a biologizing basis who might not be reached by a more matter-of-fact and critical dynamic psychobiology.

The "law of compulsion by the segment" (in states of hunger, fear, hate, love, shame, jealousy, sorrow, eroticism, etc.) to seek counter-stimulation and neutralization of its craving seems to Kempf to be "the physiology of the wish and the fundamental dynamic principle of all behavior; and not until this law and its physiology are understood and applied can normal or abnormal behavior be really understood." But why note only the segmental share in these reactions? The poor segment may suffer new therapeutic onslaughts if the doctrine is taken seriously. "Why not trim the *organ*" if its strivings are the sole or at least essential source of the tribulations of the ego? Many a surgical conscience over clitoridectomies, ovariectomies, etc., might be relieved, if the view could be proved.

The keen feeling with which Kempf, in his introduction, considered the suggestion of "neurologizing tautologies" as a nonrecognition of the intrinsic material so ably presented by him, prompts one to examine the cases in order to find whether it is life-situations and reactions described in terms of topical and diffusely emotional experiences that exhaust the description or whether there really is an appeal to *neurology* in the concrete cases. If neurologic data

exhaust any set of facts, we should accept Kempf's neurologizing—his speaking of neuroses and his emphasis on the autonomic segment—i. e., if the facts really warrant expression in neurologic rather than psychobiologic terms. As things stand, it would seem wiser to accept the facts as a psychobiologic domain, without assuming that it must first be translated into a would-be scientific theory to be scientific.

The "scientific" foundation is made up of borrowed capital, from domains in which Kempf has neither practical nor research experience nor strictly controlled training. It will not do to pass by Sherrington's views on emotions, expressed in his book (pp. 255-268), based on the fact that a dog in which appropriate spinal and vagal transections removed completely the sensation of the viscera and of all the skin and muscles behind the shoulder showed *no* obvious diminution of her emotional character: "Her anger, her joy, her disgust, and when provocation arose, her fear, remained as evident as ever," etc.; only the bristling of the coat along the back no longer occurred, while the dilatation of the pupil in anger persisted. "In view of these observations, the vasomotor theory of the production of emotion becomes, I think, untenable, also that visceral sensations or presentations are *necessary* to emotion," etc. Goltz's dog deprived of hemispheres shows that Kempf's "projicient" nervous system contains fundamental hereditarily acquired provisions, elimination of which destroyed all evidence of joy or pleasure or sex-emotions, while anger and displeasure, more definitely dependent on pain stimuli, remain at least enacted in a machine-like reflex-fashion. Hence the "autonomic segmental" function is contributory to primitive emotion, but reinforcing rather than initiating the "psychosis," after it has once been established.

It would be ungracious to take up one by one the claims of the chapter on the "physiological foundations of the personality." Many of the discussions are stimulating. But, as in so much of our pioneer work, "science" has to furnish a cloak of wholly unnecessary "authority." Let us hope that the challenge of the scientific conscience by this otherwise so meritorious work will lead to critical investigation—much as we would prefer that science might have a chance to work positively and constructively rather than be spurred on by the need of self-defense.

The formula by which Kempf measures human life does not depend on the neurologizing. It is a frank modern expression of gratification of the primary and subsidiary wishes as opposed by environmental resistance in the form of the wishes and prejudices of other people (father, mother, sister, brother, wife, husband, children, friend, priest, employer, etc.) as well as the material which wish has to work with—vocation, "disinterested" (i. e., uninterested) husband, etc. From the actual behavior (productions, many fancies, vocational pursuits, hobbies, religious and social affiliations, economic resources, addictions, hallucinations, delusions, dreams, methods of obtaining comfort, associates, etc.) and the resistance, we can disclose "what the wish or affective craving is that compels the pathological adjustment." Similarly, given the wish and the behavior, we can infer the nature of the resistance—as in the amorous wife who hallucinates sexual gratification, we "know" that the husband is indifferent, or more usually, heterosexually impotent. When the wish is ascertained and the patient recognizes and admits it as a part of his personality, the psychosis changes proportionately into an anxiety neurosis, the dissociation of the affective forces disappears through accepting the socially inferior cravings as a part of the personality (Case P. D., p. 33). The analysis

leads regressively from the conditioning of one wish to the influence of an earlier wish, and back to the adolescent and preadolescent wishes, the influence of the family, etc.

The whole scheme is an excessively determined disavowal of hereditary disposition and of *native* disproportions of individual make-up and resultant conflict. The first occasion for a discrepancy to show becomes the "cause" of the discrepancy; and the parent is emphatically judged in terms of life and conduct and its influence on the growing child and not in terms of any irresponsible germ-plasm. It certainly is a good thing to keep these concepts fluid; but there is no reason why one should go from one extreme to the other. It is a good thing to realize that the parents who want to own their children body and soul have, to say the least, not a modern philosophy; it may even cause much well-deserved heartburn to recognize the devastating influence of marital disharmony and too little and too much love and the base motives in much of the love. It might, however, make some innocent parents turn in their graves and some live ones go into depression, while, no doubt, *some* more healthy-minded ones may well incorporate some modern ideas into the code of life of the present day and of the future generations. The picture of direct retribution of fate is dramatically formulated in the legend to Michael Angelo's *Pietà*:

"The loving son of the too-devoted mother becomes the instinctive rival of the father. If he is unjust to wife and son, the son develops parricidal impulses. If he is unjust to the son and the mother is loyal to her husband, the son tends to become a wandering hero (hobo). If he is dominated by a severe, just father and pitied by a timid mother, he becomes crucified and sacrifices himself to his father's glory and potency. He often 'dies,' descends into the hell of invalidism and infantism, and is nursed and petted by the mother. Often before the sacrificial regression, he seeks a mother substitute in Magdalen, the prostitute, who having a reciprocal father attachment sympathizes with him and often marries him."

In order to make this improved moralizing and casuistry acceptable, Kempf has found himself conditioned to invent his "scientific" background. We trust that the psychobiologic formulation will be able to do justice to all the facts of experience without doing violence to both neurology and psychology. It is not the theory but the admirable vital and dynamic frankness of the author's own experience and fervor that make Kempf's book a contribution which deserves frank admiration and careful study by the student, *provided that the student knows how to preserve his or her own balance of observation through work with actual patients*. He certainly has the courage of talking the language of life. As a text or as a guide professedly written "for the professional student of human behavior who must have an unprejudiced insight into human nature in order to deal justly and intelligently with problems of abnormal behavior as they are brought to the physician, rectory, police courts, prisons and asylums, and the directors of schools and colleges, and the commanders of military and naval organizations," it offers stimulation and a mass of liberating material, which, however, should be assimilated together with personal work done under competent supervision lest a doubtful perspective and personal interpretation by the untrained and unchecked beginner might discredit the good motives and intentions. In view of the highly debatable character of the pictorial policy of the book and of its theoretical claims, it is a pity that the author furnishes the material for the published advertisements

of the book to too wide a circle of prospective beneficiaries. The failure to divide it into a book for the student and into one for a wider public may carry its own antidote.

There are many features in Kempf's presentation that go against one's judgment and against one's grain. Yet the frank and direct way, always headed for concrete situations and concrete reactions in principle at least, makes one return to the material, if it were only for the intensely dynamic point of view. Growing success and growing responsibilities will tone down many overarduous places. Whether the author will ever make any compromise with those whom he systematically shuts out from his consideration may well be doubtful, and it will not matter very much. Psychopathology is and will for some time be a rather individual affair. We have many individual brands—from Forel's hypnotism psychology, through Janet's dissociations, Sidis' hypnoidization and fear pathology, Prince's multiple personalities, Freud's elaborate analytic system, Jung's analytic constructive vision, the workers on symbolism, etc., the nosologists like Kraepelin, to the more pragmatic workers with the facts as we find them; and Kempf's may well be called the most concretely expressed of the more complex formulations and therefore very suggestive and stimulating. He is so intensely alive with what he sees and feels and works with that we may well wish him a rich experience with which to supplement what we still feel most lacking in his analysis of the "Yellow-Jacket" and throughout his discussions: a more restful view of possibilities for a species which does not have to *produce* the projicient nervous system, but receive it largely preformed and available for an existence in which the autonomic strivings have a more incidental rather than superbly dominating place. The fervent adolescent and early adult period with the sign of strife and struggle and resentment of compulsion and its desire to use compulsion, is a stage much less turbulent in most persons, psychopaths included, and the curative work can operate with a philosophy of greater stability to greater advantage than one would infer from the intensely aggressive temper of the accounts. Perhaps we are not so far from the time when men of the most different temperaments can receive and accept training in all the fundamental lines of neuropsychiatry without any morbid fear of being stunted or without having to go too exclusively their own way? For any such product of the future, Kempf can be sure to be one of the valuable contributors and sources of stimulation with facts and contrasts.

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